

# Anaesthesia

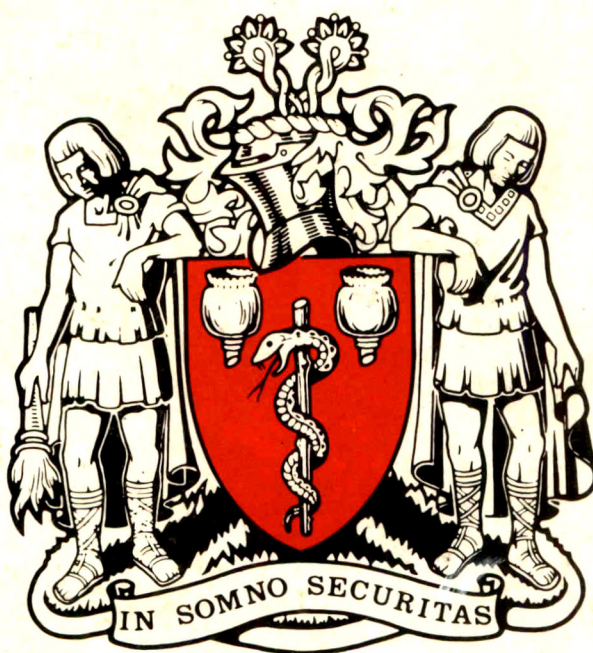
Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 46 Number 1 January 1991

CUK-402485.16-P029535

JCM

16



The Association of Anaesthetists of Great Britain and Ireland  
*Patron: HRH The Princess Margaret, Countess of Snowdon*

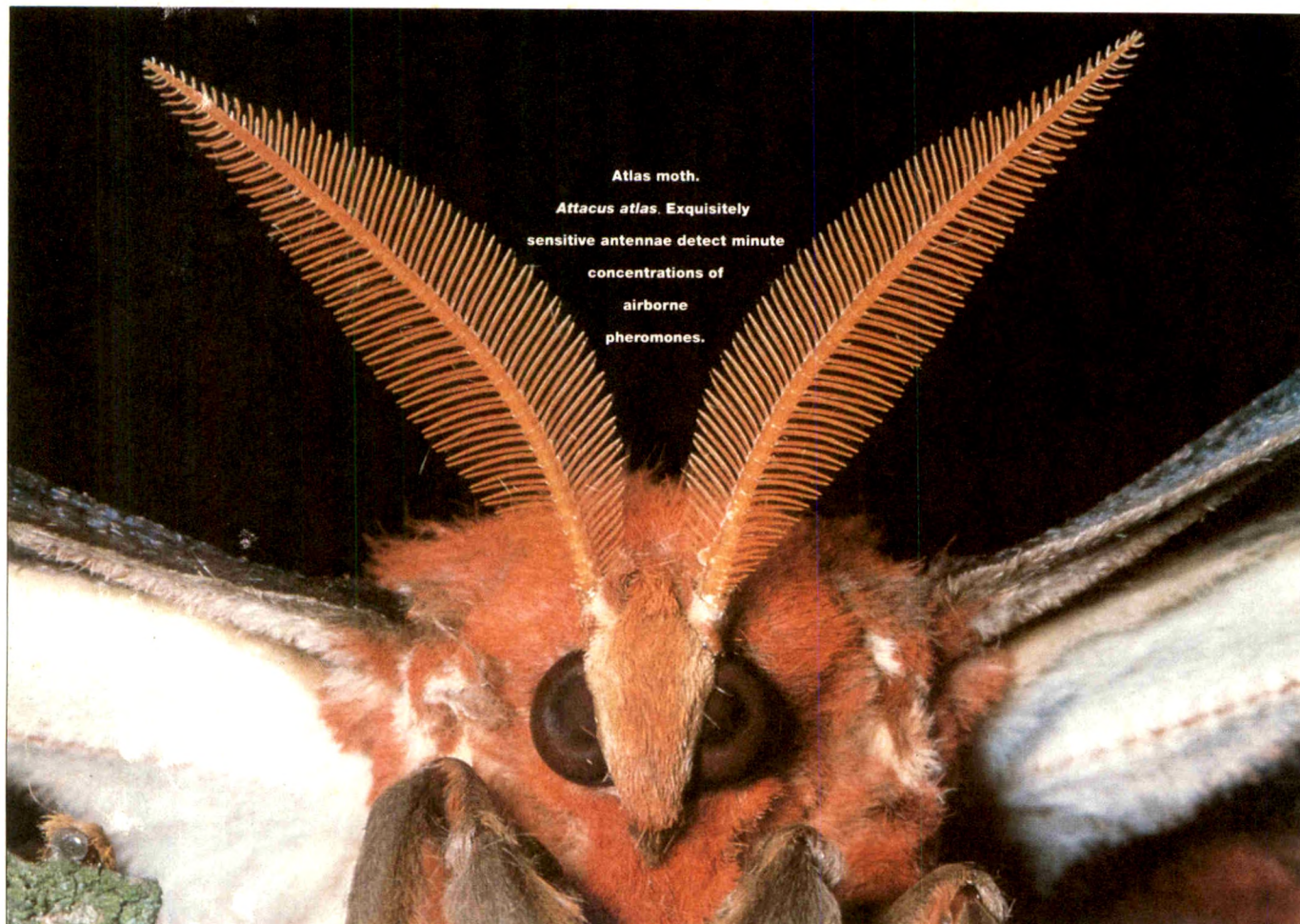


Published for the Association by  
Academic Press Grune & Stratton  
London San Diego New York Boston  
Sydney Tokyo Toronto



ISSN 0003-2409





Atlas moth.  
*Attacus atlas*. Exquisitely  
 sensitive antennae detect minute  
 concentrations of  
 airborne  
 pheromones.

## Is this an anaesthetist's role model?

*An anaesthetist, like a moth,  
 develops a sensitivity bordering on "sixth sense";  
 mental antennae attuned to every  
 requirement of the patient.*

P 24,535

P 24,535

**Diprivan**▼  
 propofol  
 i.v. anaesthesia

### **P R E S C R I B I N G   N O T E S**

▼Special reporting to the CSM required.

Consult data sheet before prescribing.

**Use:** Induction and maintenance of general anaesthesia for surgical procedures lasting up to one hour.

**Presentation:** Ready to use, isonic, aqueous emulsion containing 10mg/ml propofol in a vehicle containing glycerol oil and purified egg phosphatide.

**Dosage and administration:** **Induction:** Titrate against response using approximately 4ml every 10 seconds in healthy adults and 2ml every 10 seconds in patients of ASA grades 2 and 4. Patients under 55 years are likely to require 2.0 to 2.5mg/kg, older patients may require approximately 20% less.

**Maintenance:** Usually 0.1 to 0.2mg/kg/min (6 to 12mg/kg/hr). Continuous infusion may require slightly higher rates for 10 to 20 minutes after induction. The infusion can be used undiluted or diluted with 5% Dextrose (intravenous infusion BPI). Alternatively repeat bolus injections of 25 to 50mg (2.5 to 5.0ml) may be administered according to clinical need.



**Paediatric Use:** No experience in children or in mothers who are breast feeding.

**Contraindications:** Allergy to 'Diprivan'.

**Precautions:** Ampoules and vials should be inspected before use for particulate matter and discolouration. Aseptic technique should be followed throughout its handling; administration should commence without delay once drawn into the syringe or giving set. Discard any unused 'Diprivan' after single patient use. Do not mix prior to administration with other agents or infusion fluids other than Dextrose 5%, such dilutions should be prepared immediately before administration. Hypotension and transient apnoea may occur during induction. Occasionally, hypotension may require use of i.v. fluids and a lower rate of administration during maintenance. Apply caution in cardiac, respiratory, renal or hepatic impairment, epilepsy, in hypovolaemic or debilitated patients, and in disorders of fat metabolism or conditions where lipid emulsions should be used cautiously. Bradycardia may occur due to the lack of vagolytic activity of 'Diprivan' and administration of an anticholinergic should be considered particularly if vagal

tone is dominant. Do not use in pregnancy except for termination. Discharge after general anaesthesia - allow adequate time for full recovery.

**Side effects:** Epileptiform movements, including convulsions and opisthotonus, have occurred rarely in a temporal relationship to 'Diprivan'. Nausea, vomiting and headache in a small proportion of patients. Very rarely, clinical features of anaphylaxis, which may include bronchospasm and erythema accompanied by hypotension, have been reported with 'Diprivan'. Pain on injection in a proportion of patients. Discolouration of urine, venous sequelae and fever are rare. Minimal evidence of excitation on induction.

**Product licence number:** (29/0190). **Basic NHS cost:** £3.98 per 20ml ampoule, £9.95 per 50ml infusion vial.

'Diprivan' is a trademark.

Further information is available from ICI Pharmaceuticals (UK), Southbank, Alderley Park, Macclesfield, Cheshire SK10 4TF.



# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Editor M. MORGAN

Assistant Editors R. GREENBAUM, A. R. AITKENHEAD, D. E. F. NEWTON, R. A. MASON, M. HARMER

## Editorial Board

Chairman The President of the Association P. J. F. BASKETT

Members A. M. ROLLIN, C. A. B. MCLAREN and a representative of the Junior Anaesthetists' Group

## Ex officio

The Honorary Treasurer of the Association W. R. MACRAE

The Honorary Secretary of the Association J. E. CHARLTON

The Immediate Past Honorary Secretary of the Association W. L. M. BAIRD

DIANE WINTERBURN, JANET SPRINGER, Reference librarians

*Address for literary contributions* Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK [telephone 081-740 3290].

Neither the Editor nor the Association of Anaesthetists of Great Britain and Ireland is in any way responsible for the statements made or the views expressed by contributors.

Investigations performed on man must conform to appropriate ethical standards including voluntary, informed consent and acceptance by an ethics committee.

A written statement of this acceptance may be requested by the Editor.

ANAESTHESIA: ISSN 0003-2409, Volume 46 1991, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. **All advertising** enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293).

Annual subscription price including postage: £132 UK and US \$244 overseas. Personal subscription rate including postage: \$99. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel. 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

*Anaesthesia* is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

© 1991 The Association of Anaesthetists of Great Britain and Ireland. The appearance of the code at the bottom of the first page of a paper in this journal indicates the copyright owner's consent that copies of the paper may be made for personal or internal use, or for the personal or internal use of specific clients in the USA. This consent is given on the condition, within the USA, that the copier pay the stated per-copy fee through the Copyright Clearance Center Inc., 27 Congress Street, Salem, MA 01970, USA for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to other kinds of copying, such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, for resale or for copying or distributing copies outside the USA.

Origination by BPCC Whitefriars Ltd, Tunbridge Wells, and printed and bound in Great Britain by BPCC Wheatons Ltd, Exeter.



# UNIVERSITY OF LIVERPOOL

## DEPARTMENT OF ANAESTHESIA

### PART III F.C. ANAES. COURSE : APRIL 8th-13th 1991

A six-day course consisting of lectures, MCQ, essay and viva examinations will be held in this department, suitable for those preparing for this examination in May 1991.

ORGANISER — DR. J.M. HUNTER

Fee — £175.00

#### Further details from:

The Postgraduate Secretary,  
University Department of Anaesthesia,  
Royal Liverpool Hospital,  
Prescot Street,  
P.O. Box 147,  
LIVERPOOL L69 3BX.

### 11th Myron B. Laver

### International Postgraduate Course

### «PAIN»

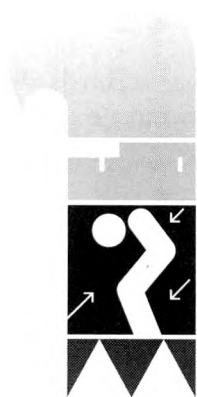
Department of Anaesthesia, University of Basel,  
Switzerland, March 22-23, 1991

#### Participating Faculty:

K J S Anand,  
Children's Hospital,  
Boston, USA  
Michael J Cousins,  
University of Sydney  
at the Royal North Shore Hospital,  
St. Leonards, Australia  
Benjamin G Covino,  
Brigham and Women's Hospital,  
Harvard Medical School,  
Boston, USA  
Henry J McQuay,  
Oxford Regional Pain Relief Unit,  
Abingdon Hospital,  
Abingdon, Oxon, GB  
Terence M Murphy,  
School of Medicine,  
University of Washington,  
Seattle, USA

P Prithvi Raj,  
Health Science Center,  
University of Texas,  
Houston, USA  
L Brian Ready,  
School of Medicine,  
University of Washington,  
Seattle, USA  
Donald C Tyler,  
Children's Hospital  
and Medical Center,  
University of Washington,  
Seattle, USA  
Patrick D Wall,  
Middlesex School of Medicine,  
University College London,  
London, GB

For further information contact:  
The Secretariat, Department of Anaesthesia,  
University of Basel/Kantonsspital,  
CH-4031 Basel, Switzerland  
Fax 061/25 60 22





# The World's Premier Medical Publisher

## EPIDURAL & SPINAL BLOCKADE IN OBSTETRICS

Edited by

**Felicity Reynolds**, *Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.*

This book has evolved from the Obstetric Anaesthetist's Association meeting in the UK in September 1989. Unlike other books of proceedings however, the well-known contributors were invited not only to speak but also to provide a chapter reviewing their subject, prepared especially for the book.

Edited by Dr. Reynolds, the result is an interesting blend of a review of the last ten year's developments coupled with presentations of current research and coloured throughout with stimulating, amusing discussion.

- Includes unique illustrations, using state-of-the-art imaging techniques, of the anatomy of the epidural space.
- Features excellent reviews of the rapidly expanding fields of Caesarean section and the use of spinal opioids.

The closing section, The Recipient, discusses the effects that conduction blocks may have on the baby, and reviews the lay literature covering consumer's views of epidural practice.

### CONTENTS

The Epidural Space: Anatomy and Approach • Indications and Contraindications to Epidural Blockade • Test Doses • Top-ups Versus Continuous Infusion • Management of the Second Stage of Labour • Local Anaesthetic Toxicity • Taps and Patches • Neurological Complications of Epidural and Spinal Blockade • Regional or General Anaesthesia for Emergency Caesarean Section • Spinal and Epidural Blockade • Chairman's Responses • Epidural Opioids in Labour • Epidural Opioids for Caesarean Section • The Effects of Regional Analgesia • Public Opinions • Summary and Conclusions • Index.

**0 7020 1401 X ca 250pp Ills Pb**  
**August 1990 Baillière Tindall £17.50**

## ESSENTIALS OF ANESTHESIOLOGY 2/e

**David C. Chung**, *Consultant, Dept Anaesthesiology, The Mississauga Hospital, Ontario, Canada;*

**& Arthur M. Lam**, *Assoc. Prof., Dept Anaesthesiology, Harborview Medical Center, Univ. Washington, Seattle, USA.*

A primer in anaesthesia written for those about to specialise in the field, this book provides concise, yet thorough coverage of the scientific basis and the principles of safe practice. All chapters have been revised to incorporate new drugs, equipment and medical conditions.

Includes:

- Revised and expanded section on management of postoperative pain
- New section on quality assurance and risk management
- New chapter on AIDS and the anaesthetist
- New section on autologous blood transfusions

**0 7216 3084 7 304pp 50 ills SpPb**  
**1989 W.B. Saunders £15.50**

## TEXTBOOK OF PAEDIATRIC ANAESTHETIC PRACTICE

**E. Sumner & D. Hatch**

**0 7020 1336 6 560pp 103 ills Hb**  
**1989 Baillière Tindall £37.50**

## INTENSIVE CARE: A Concise Textbook

**C.J. Hinds**

**0 7020 1150 9 387pp 109 ills Pb**  
**1986 Baillière Tindall £15.95**

## OBSTETRIC ANAESTHESIA & ANALGESIA 3/e

**D.D. Moir & J. Thorburn**

**0 7020 1154 1 390pp Ills Hb**  
**1986 Baillière Tindall £23.00**

## DIFFICULTIES IN TRACHEAL INTUBATION

**I.P. Latta & M. Rosen**

**0 7020 1096 0 192pp 150 ills Hb**  
**1985 Baillière Tindall £19.95**

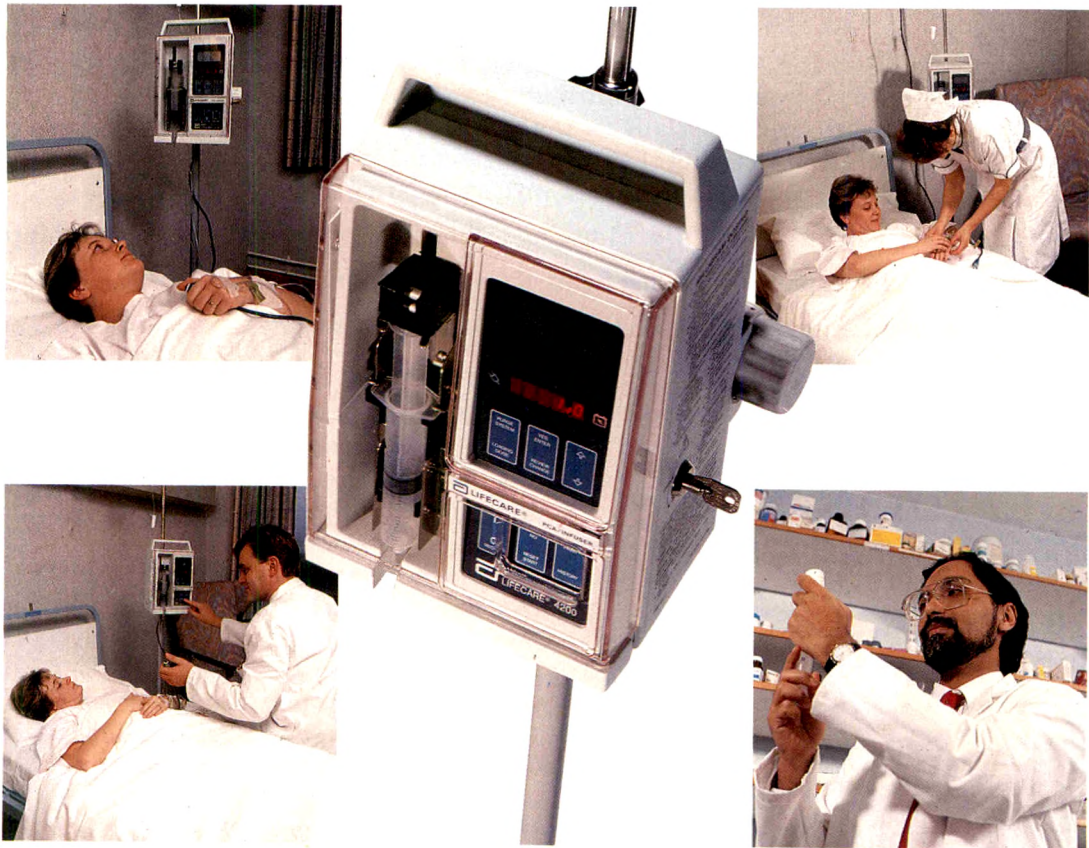
**W.B. Saunders & Baillière Tindall**

**HARCOURT BRACE JOVANOVICH**

Available from your bookseller. For more information please contact: The Marketing Dept, Harcourt Brace Jovanovich, 24-28 Oval Rd, London NW1 7DX, UK.  
OR phone 081 300 3322  
Prices and specifications may vary without notice.



# ABBOTT PATIENT CONTROLLED ANALGESIA



## Allowing the patient membership of the pain management team...

- + Allows the patient to control their pain.
- + Allows the nurse time to manage the patient.
- + Allows the doctor to establish the dose.
- + Allows the pharmacist to monitor the drug.

If you would like to offer extra membership to your pain management team, please ask for more information from:

**ABBOTT**

**ABBOTT LABORATORIES LIMITED**  
Queenborough, Kent ME11 5EL  
Tel: (0795) 580099 Fax: (0795) 580404

**PCA**

## Editorial

### Tributes and tribulations

The first issue of *Anaesthesia* in 1991 sees the journal under the control of a new editor, the fifth since it first appeared in 1946. It is also the first issue for 17 years with which John Lunn has not been involved, initially as an assistant editor and for the last 10 years as editor. In those years the journal has expanded and flourished under his guidance; he now has more time to devote his considerable energies to the vital topic of audit and in particular the Confidential Enquiries into Perioperative Deaths.

Other people too are ending their association with the journal. For 25 years Dr Leon Kaufman has compiled the Anaesthetic Literature section with much effort and enthusiasm. This service has now been superseded by electronic retrieval systems which are available in the majority of libraries; this will allow more space for scientific articles. Dr Donald Bateman has for many years acted as honorary proof reader of copy just prior to going to press. He is also retiring and it has been decided that he should not be replaced. Dr Cyril Scurr is also leaving having served on the editorial board since 1983; his wise counsel will be greatly missed. We thank these three hard working doctors for giving their time so generously over the years, and wish them well in the future.

It is little known that the references of all articles published in *Anaesthesia* are checked by a professional librarian, which as far as I know is the only scientific journal offering this service. It is extremely rare to receive an article with impeccable references and indeed your editor has been horrified at the number of mistakes that have been found in the references of articles that he has submitted to this journal in the past. Miss Jacqueline Welch, the senior librarian is now giving up after 9 years to become personal assistant to a European MP. The speed with which she returned references and the detail with which they were checked, even to missing hyphens, was remarkable; even references in foreign languages were translated. She will be sorely missed by the editor and his assistants. We wish her well in her new career.

A new editor is expected to make some statement as to any change in editorial policy. This, however, remains basically as before, namely to report and support the advancement of clinical and academic anaesthesia and to encourage you, the readers, to publish the findings of your investigations. Every editor wishes to publish exciting and novel original work, but we can only do this if we receive such papers. Review articles are always welcome, but they should be written from a background of experience and authority. The Forum section was originally introduced for publication of preliminary communications, short papers, or those which perhaps did not fulfil the criteria of a properly controlled trial, or in which there were no comparisons, yet which were interesting enough to warrant publication. Indeed, some authors submit articles specifically for Forum. It is intended that this shall continue. Case reports with a specific anaesthetic interest presenting

problems of management will also be welcome, but not those rare cases which just happen to include an anaesthetic as part of the treatment. The journal has always had a thriving correspondence section and long may it continue. It will also be our policy to attempt to publish articles as soon as possible, but with the amount of material that is received, some delay is inevitable.

And now to the problem of rejection. The large volume of material reaching the editor's desk is very welcome, but it does make manuscript selection much more difficult. We try to publish something that will be of interest to everyone in each issue, but this is really an impossible task and everyone cannot always be satisfied. About 25-30% of articles received are manifestly unsuitable for the journal, whereas about 5-10% are immediately acceptable. *Anaesthesia* is a peer review journal which, despite the views of some to the contrary, is still the best way of assessing a manuscript's suitability for publication. The editor is indeed fortunate in having five experienced and dedicated assistants, namely Professor Aitkenhead and Drs Greenbaum, Newton, Mason and Harmer whose opinions would be valued by the editor of any anaesthetic or related journal.

Nevertheless, manuscripts are often reviewed by assessors outside the Editorial Board. Attempts have been made in the past for articles to be assessed on a double-blind basis, but this was not acceptable to the reviewers who objected to the suggestion that they could be biased if they knew the origin of the manuscript.

Articles are accepted for publication on merit and not on literary style. However, concisely written, well presented papers can be handled more rapidly by reviewers and can be subedited and prepared for press in a matter of minutes. Articles that do not comply with the style of the journal can take days or weeks to prepare, since each item that does not conform has to be altered. This work is done by the assistant editors at night and at weekends, backed up by the efficient Sue Sharples at Academic Press. Authors are requested to be vigilant over unnecessary length and also to consult the notice to contributors that is found in each issue of the journal.

Writing rejection letters is one of the most difficult editorial tasks and it is hard to find the right words to cushion the blow of having an article turned down; the new editor is no stranger to the receipt of such letters. The editor tries to point out where the reviewers think that the work has gone wrong and on occasion will suggest changes to the manuscript and resubmission. Most accept receipt of a rejection letter philosophically, but sometimes the response can be alarming and even irrational. It is doubtful, however, if any editor will ever be able to repeat the eloquence of a rejection letter that was reputedly from a Chinese economics journal and of which I was recently reminded.<sup>1</sup> 'We have read your manuscript with boundless delight. If we were to publish your paper, it would be impossible for us to publish any work of a lower standard. And as it is unthinkable that, in the next thousand years we shall see its equal, we are,



to our regret, compelled to return your divine composition and to beg you a thousand times to overlook our short sight and timidity'.

It is a privilege to be editor of a journal such as *Anaesthesia* and it is with some trepidation that I embark on this task in the footsteps of such illustrious predecessors, but I do so in the knowledge that I have the full backing of the Association of Anaesthetists of

Great Britain and Ireland and five excellent assistant editors.

M. MORGAN  
*Editor*

#### Reference

1. TURNER T. Writing for the Biochemical Journal. *Biochemist* 1990; 12: 13-4.

#### Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; 1: 432-5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

## A comparison of the performance of 20 pulse oximeters under conditions of poor perfusion

D. G. CLAYTON, R. K. WEBB, A. C. RALSTON, D. DUTHIE AND W. B. RUNCIMAN

### Summary

*The performance of 20 pulse oximeters with finger probes was evaluated by comparison of their readings with directly measured arterial blood oxygen saturations. The samples were taken from patients who had undergone cardiac surgery under hypothermic cardiopulmonary bypass and had poor peripheral perfusion. The mean difference (bias, accuracy), standard deviation (precision) and drop-out rate for each pulse oximeter was determined. An overall ranking of performance of each pulse oximeter was calculated using five criteria (accuracy, precision, number of readings within 3% of standard, percentage of readings given within 3% of standard, expected overread limit in 95% of cases). Two pulse oximeters achieved a combination of accuracy and precision such that 95% of measurements would be expected to be within 4% of the co-oximeter value; these two also had the lowest drop-out rate.*

### Key words

*Equipment; pulse oximetry.  
Measurement.*

Pulse oximeters are widely used during anaesthesia in the peri-operative period, and in critically ill patients. Pulse oximetry has developed rapidly over the last 5 years; its history, basic principles, applications and limitations have been the subject of three recent comprehensive reviews.<sup>1–3</sup> The technique has come into wide clinical use so it is important to examine circumstances wherein its reliability may be questioned. Performance under conditions of poor perfusion is one such circumstance.

Pulse oximeters require adequate plethysmographic pulsations to allow them to distinguish arterial blood light absorption from background venous blood and tissue light absorption. Only 1–5% of the total signal is processed to calculate the haemoglobin saturation.<sup>4</sup> Hypothermia, vasoconstriction and low cardiac output may impair peripheral perfusion, and thus plethysmographic pulsation and the portion of total signal used to detect haemoglobin saturation.

Other attempts have been made to study the performance of pulse oximeters under conditions of poor peripheral perfusion. Morris produced venous congestion using a pneumatic tourniquet and took this to represent poor peripheral perfusion.<sup>5</sup> Fifteen oximeters were compared under these conditions, but the validity of this technique was questioned.<sup>6</sup> Superficial cooling was also used to reduce peripheral perfusion.<sup>7</sup> However, in both these

studies observations were not compared with the standard reference of co-oximetry.

This study was designed to overcome some of these deficiencies by comparing the performance of 20 oximeters with *in vitro* measurements of haemoglobin saturation using a four wavelength co-oximeter in patients after cardiac surgery involving cardiopulmonary bypass and hypothermia. Such patients have been shown by Kuttilla to have poor peripheral perfusion.<sup>8</sup> In Kuttilla's studies peripheral perfusion measured by skin red cell flux did not return to normal until rectal temperature had increased to 37.5°C. Reperfusion of the peripheral vascular bed was delayed until 7 hours after surgery.<sup>8</sup>

### Methods

#### *Patient selection*

The study was approved by the local Human Ethics Committee. Adult patients were studied 30 minutes to 2 hours after transfer from the operating theatre to the cardiac surgery recovery ward, and all had undergone surgery involving cardiopulmonary bypass with hypothermia to 25–31°C. All patients were sedated and were undergoing intermittent positive pressure ventilation of the lungs at the time of the study.

D.G. Clayton, BSc, MB, BS, FFARCS, R.K. Webb, MB, BS, FFARCS, Senior Staff Specialists, A.C. Ralston, BSc, BAplSc, Medical Physicist, D. Duthie, MB, BCh, FFARCS, Senior Registrar, Professor W.B. Runciman, BSc(Med), MBBCh, FFARCS, PhD, Head of Department, University of Adelaide, Australian Patient Safety Foundation, GPO Box 400, Adelaide, South Australia 5001.

Accepted 15 July 1990.



### Observations

The age, sex, operation, rectal temperature, systolic, diastolic and mean blood pressures and drug therapies of each patient were recorded.

The performance of 20 pulse oximeters was studied. They were supplied by their manufacturers and were checked for compliance with the Australian Standard 3200-1986 on electromedical equipment for use in patient care areas.

Pulse oximeter probes were placed on patients' fingers according to the manufacturers' instructions. Systemic arterial pressures were monitored directly and continuously through radial artery cannulae. Pulse oximeter probes were applied only to fingers on the arm not bearing the arterial cannula. Probes were not applied to the thumb. The finger used for each manufacturer's probe was varied in a predetermined fashion to ensure that each probe was placed an equal number of times on each of the fingers used, and so that any possible systematic interference from adjacent probes was eliminated. A two-dimensional matrix was constructed after allocating a number to each oximeter and a letter to each finger so that it could be visually checked that these requirements were met. All probes were covered by opaque rubber sleeves to reduce the likelihood of cross-over radiation, and were allowed to stabilise for 3 minutes before observations were made.

The haemoglobin saturation and pulse rate displayed on oximeters were recorded. A 2-ml sample of heparinised arterial blood was taken at the same time from the indwelling radial artery cannula. It was noted when pulse oximeters failed to give readings of haemoglobin saturation.

The 20 pulse oximeters were arranged on three trolleys in the groups given below.

- Group 1 *Datex Satlite* (Datex Instrumentarium Corp. Helsinki, Finland)  
*Invivo 4500* (Invivo Research Laboratories, Broken Arrow, Oklahoma, USA)  
*Nellcor N-200* (Nellcor Incorporated, Hayward, California, USA)  
*Novamatrix 505* (Medical Systems Inc., Wallingford, Connecticut, USA)  
*Ohmeda Biox 3740* (Ohmeda, Boulder, Colorado, USA)  
*Radiometer Oximeter* (Radiometer A/S, Copenhagen, Denmark)
- Group 2 *Datascope Accusat* (Datascope Corp. Paramus, New Jersey, USA)  
*Ohmeda Biox 3700* (Ohmeda, Boulder, Colorado, USA)  
*Nonin 8604D* (Nonin Medical, Inc., Plymouth, Minnesota, USA)  
*Physio-Control 1600* (Physio Control, Redmond, Washington, USA)  
*Sensormedics Oxyshuttle* (SensorMedics Corporation, Anaheim, California, USA)  
*Simed S-100* (Simed Corporation, Bothell, Washington, USA)  
*Spectramed Pulsat* (Spectramed Inc., Oxnard, California, USA)
- Group 3 *Biochem Microspan 3040* (Biochem International Inc., Waukesha, Wisconsin, USA)

*Criticare CSI 503* (Criticare Systems, Inc., Milwaukee, Wisconsin, USA)

*Criticare CSI 504* (Criticare Systems, Inc., Milwaukee, USA)

*Engstrom Eos* (Gambro Engstrom AB, Bromma, Sweden)

*Kontron 7840* (Kontron Instruments, Watford, UK)

*Minolta Pulsox 7* (Minolta Camera Co. Ltd., Osaka, Japan)

*Pulsemate Colin BX-5* (Nippon Colin Co. Ltd., Hayashi, Japan)

Each of 40 patients was tested by every oximeter on one of these trolleys. There were more oximeters than fingers available for each test, so haemoglobin saturation was measured twice in each patient by pulse oximetry and arterial blood co-oximetry. Each pulse oximeter was tested once on 40 different patients. The oximeters were alternatively tested on the first or second run for each patient so that the possibility of any systematic bias against a particular oximeter was eliminated. The three groups of oximeters were tested sequentially in the order given.

### Co-oximetry and arterial blood gas analysis

Arterial blood samples were collected anaerobically, stored on ice and analysed by co-oximeter (IL 482, Instrumentation Laboratory, Lexington, Massachusetts, USA). Samples were analysed within one hour of collection. The co-oximeter was calibrated weekly against the manufacturer's 'Cal Dye' solutions of known haemoglobin concentration. Three test solutions of preserved blood of known saturations supplied by Instrumentation Laboratories were analysed daily to confirm that the performance of the machine was within specification. All procedures were performed according to the manufacturer's instructions.

### Statistical analysis

Descriptive statistics were calculated for all pulse oximeters studied and for the three groups of 40 patients tested using the three groups of pulse oximeters. Comparisons between patient groups were made using a commercial statistics package (Statgraphics, Statistical Graphics Corporation, Rockville, Maryland, USA) and analysis of variance (Scheffe method for parametric data and Kruskal-Wallis method for nonparametric data). Statistical significance was accepted when  $p < 0.05$ .

The co-oximeter measurement was subtracted from the corresponding haemoglobin saturation displayed by each pulse oximeter to give the difference (bias) of pulse oximeter from co-oximeter measurement. The mean of these differences was taken as the mean bias (accuracy). Precision was taken to be one standard deviation of the differences between the pulse oximeter and the co-oximeter.

### Results

One hundred and twenty patients were studied. The means and standard deviations of the measurements made on all 120 patients are given in Table 1. The means and standard deviations of the 40 observations made when each group was tested appear in Table 2. Analysis of variance showed no significant differences between the groups with respect

**Table 1.** Means and standard deviations of measurements made on all patients ( $n=120$ ). Co-oximetry was performed twice on each patient ( $n=240$ ).

Measurement	Mean	SD
Age (years)	60	9.4
Rectal temperature ( $^{\circ}\text{C}$ )	35.1	0.75
Systolic arterial pressure (mmHg)	114	15.4
Diastolic arterial pressure (mmHg)	59	9.4
Pulse pressure (mmHg)	55.4	15.6
Heart rate (beats/minute)	96	16.2
Haemoglobin saturation %	96.7	1.7
Carboxyhaemoglobin %	2.0	0.54
Methaemoglobin %	0.5	0.39

to age, rectal temperature, heart rate, co-oximeter oxygen saturation, carboxyhaemoglobin, methaemoglobin and blood gas machine bicarbonate and oxygen saturation. Differences between patients with respect to systolic and diastolic arterial pressures were significant at the 5% level. However, differences in pulse pressures between the three groups were not significant. Given that the mean systolic and diastolic blood pressure were all within the normal clinical range, and that there was no difference in pulse pressure, no bias toward any particular trolley should have been caused by the small differences in systolic and diastolic pressures between the trolleys.

**Table 2.** Means and standard deviation on three groups of 40 patients tested sequentially using three groups of oximeters. Analysis of variance (ANOVA) at 2117 degrees of freedom: F statistic and significance level,  $p$ . (\*= $p<0.05$ )

Measurement	Mean	SD	
Age (years)			
Group 1	60.5	8.6	ANOVA
Group 2	61.2	10.1	$F=0.25$
Group 3	59.7	9.6	$p<0.8$
Rectal temperature ( $^{\circ}\text{C}$ )			
Group 1	35.1	0.85	ANOVA
Group 2	35.1	0.73	$F=0.29$
Group 3	35.2	0.66	$p<0.8$
Systolic arterial pressure (mmHg)			
Group 1	118	15.9	ANOVA*
Group 2	109	14.4	$F=3.96$
Group 3	116	14.3	$p<0.02$
Diastolic arterial pressure (mmHg)			
Group 1	60.2	10.0	ANOVA*
Group 2	55.9	7.6	$F=3.29$
Group 3	60.8	10.1	$p<0.04$
Pulse pressure (mmHg)			
Group 1	57.4	16.9	ANOVA
Group 2	53.0	14.0	$F=0.79$
Group 3	55.7	16.2	$p<0.45$
Heart rate (beats/minute)			
Group 1	96.3	13.3	ANOVA
Group 2	94.5	19.4	$F=0.36$
Group 3	97.9	15.6	$p<0.7$
Co-oximeter			
Haemoglobin saturation (%)			
Group 1	97.0	0.91	ANOVA
Group 2	96.5	1.59	$F=1.31$
Group 3	96.5	2.14	$p<0.3$
Carboxyhaemoglobin (%)			
Group 1	2.0	0.44	ANOVA
Group 2	2.1	0.52	$F=0.11$
Group 3	2.0	0.65	$p<0.9$
Methaemoglobin (%)			
Group 1	0.48	0.31	ANOVA
Group 2	0.48	0.57	$F=2.25$
Group 3	0.66	0.35	$p<0.1$

Most of the oximeters failed to give a reading on a number of patients as a result of poor signal quality. This drop-out rate was expressed as the percentage of tests for which a pulse oximeter gave no reading because of low quality signals. The ability to recognise a weak or very noisy waveform which could cause an erroneous saturation reading is a safety feature of all the models used in this trial. Instead of showing saturation the oximeter display is blank, or it may display a flashing value or a message which indicates a poor quality signal. Five of the 20 oximeters tested gave readings on all 40 patients (Fig. 1). The number of readings within 2% of the co-oximeter ranged from 11 to 32, while the number of readings within 3% of the co-oximeter ranged from 20 to 40 (Fig. 1). The number of readings within 3% of the co-oximeter reading was expressed as a percentage of the total readings and ranged from 100% down to 57% (Table 3).

The mean difference (accuracy) of the pulse oximeters differed by 0.1 to 4.5% haemoglobin saturation from co-oximeter values. Pulse oximeters varied from underestimating haemoglobin saturation by a mean of 4.5 % to overestimating it by a mean of 2.7% (Fig. 2). Sixteen pulse oximeters tended to overestimate haemoglobin saturation and four underestimated it. Differences in precision between oximeters that overread and underread haemoglobin saturation reached statistical significance. The precision, or standard deviation of the differences between pulse oximeter and co-oximeter, varied from 0.96% to 5.78% (Fig. 3).

We can determine the 95% confidence limits ( $\text{mean} \pm 1.96 \times \text{SD}$ ) within which we would expect 95% of the readings of the pulse oximeters to fall by use of the individual values for bias and precision. These are displayed in Figure 4. The upper and lower limits of these ranges are not equidistant from zero.

There was no significant difference between the three trolleys with respect to ranking for accuracy, precision, drop-out rate,  $\pm 3\%$  values,  $\pm 2\%$  values, 95% positive limits or final overall combined ranking using Kruskal-Wallis analysis of variance.

## Discussion

Manufacturers specify commonly that pulse oximeters have a standard deviation, when compared to a 'gold standard' of 1.5–2.5% (greater than 75% of units we tested state 2%) at saturations in the range 90–100%. One interpretation of this is that 95% of readings should be within 1.96 times the standard deviation (4% saturation in the majority) of the true value. Only two out of the 20 pulse oximeters met this criterion, with one additional oximeter meeting its manufacturer's (but not this) specification. The algorithms used to process the plethysmographic signal electronically are compared with co-oximetry data during development. Few human data of haemoglobin saturations below 70% are available and the accuracy claimed by manufacturers below 70–100% saturation is often less than 2%; these findings were confirmed in several comparative studies of oximeters,<sup>2,9</sup> and will be summarised and reviewed in the second paper of our series on potential sources of pulse oximeter error.

This study differs from previous studies in two respects. Firstly, a greater number of oximeters was available for study. Secondly, instead of investigating the oximeters on



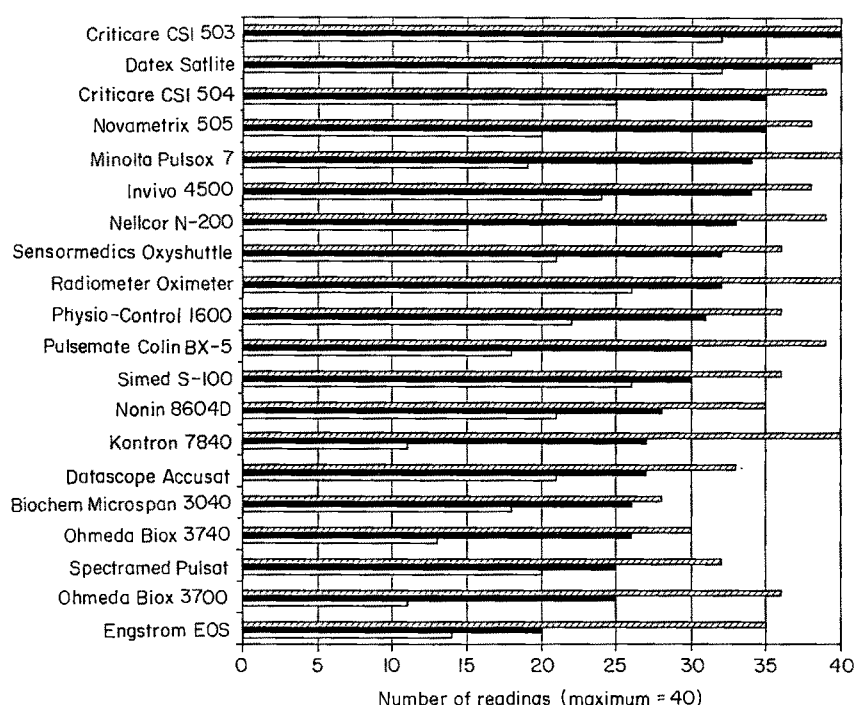


Fig. 1. The total number of readings and those within  $\pm 3$  and  $\pm 2\%$  of the co-oximeter reading. Each pulse oximeter was tested on 40 patients. ▨, total; ■,  $\pm 3\%$ ; □,  $\pm 2\%$

volunteers or warm, well-perfused patients, we examined only poorly perfused patients in the hope that this would prove to be a more discriminative model.

We have been able to verify the poor perfusion status of our patient population by comparing the drop-out rate for these patients with that for a better perfused population. Fourteen of the pulse oximeters used in this study were also used for a similar trial which involved intensive care unit patients, in which the oximeters were assessed by the same investigators using the same basic protocol described in this paper. The average drop-out rate for the intensive care unit

patients was 0.26%, whereas that in this study was 8.7%, that is 33 times higher, and reflects the relatively poor perfusion status of patients in this study.<sup>9</sup>

Morris *et al.* compared 15 oximeters under conditions of poor perfusion.<sup>5</sup> This study may be criticised on two counts. Firstly, rather than using a saturation measured by co-oximeter as their 'gold standard' they used an arbitrarily chosen oximeter on the contralateral arm. Secondly, poor peripheral perfusion was produced by occlusion using a pneumatic tourniquet. This model clearly differs from poor peripheral perfusion in the postoperative cardiac patient, and it was suggested that it represents venous occlusion.<sup>6</sup> Other workers<sup>2,7,10</sup> have examined how various oximeters perform when peripheral perfusion is reduced, but examined only small numbers of oximeters. Tremper concluded that pulse oximeters are sufficiently accurate for clinical purposes over a wide range of haemodynamic conditions, but examined only the Biox III Ohmeda oximeter.<sup>10</sup>

Wilkins *et al.* studied four pulse oximeters under conditions of venous engorgement caused by inflation of a sphygmomanometer cuff to 40 mmHg and vasoconstriction induced by placing the subject's arm in a cold water-filled plastic envelope.<sup>7</sup> They found that under both experimental conditions the detection time for induced hypoxaemia was significantly increased. They noted in addition marked differences in the oximeters tested both in their susceptibility to vasoconstriction and venous congestion and in their ability to detect desaturation. One deficiency in this study was that each oximeter acted as its own control and no reference saturation, such as that measured by a co-oximeter, was used. Thus no measure of absolute accuracy was given for the oximeters under test. The application of venous engorgement is, as in the study of Morris *et al.*,<sup>5</sup> of doubtful clinical validity.

One recent paper<sup>11</sup> examined two pulse oximeters on patients immediately after open heart surgery. The lowest

Table 3. Accuracy of pulse oximeters, ranked according to number of readings within 3% and showing ranking for number of readings within 3% of total number of readings expressed as percentage.

Oximeter	Total	±3%	Percent ±3%/total	Rank
Criticare CSI 503	40	40	100	1
Datex Satellite	40	38	95	2
Novamatrix 505	38	35	92	4
Criticare CSI 504	39	35	90	5
Invivo 4500	38	34	89	6
Minolta Pulsox 7	40	34	85	10
Nellcor N-200	39	33	85	10
Sensormedics Oxyshuttle	36	32	89	6
Radiometer Oximeter	40	32	80	14
Physio-Control 1600	36	31	89	6
Simed S-100	36	30	83	12
PulseMate Colin BX-5	39	30	77	17
Nonin 8604D	35	28	80	14
Kontron 7840	40	27	68	19
Datascope Accusat	33	27	82	13
Biochem Microspan 3040	28	26	93	3
Ohmeda Biox 3740	30	26	87	9
Spectramed Pulsat	32	25	78	16
Ohmeda Biox 3700	36	25	69	18
Engstrom Eos	35	20	57	20

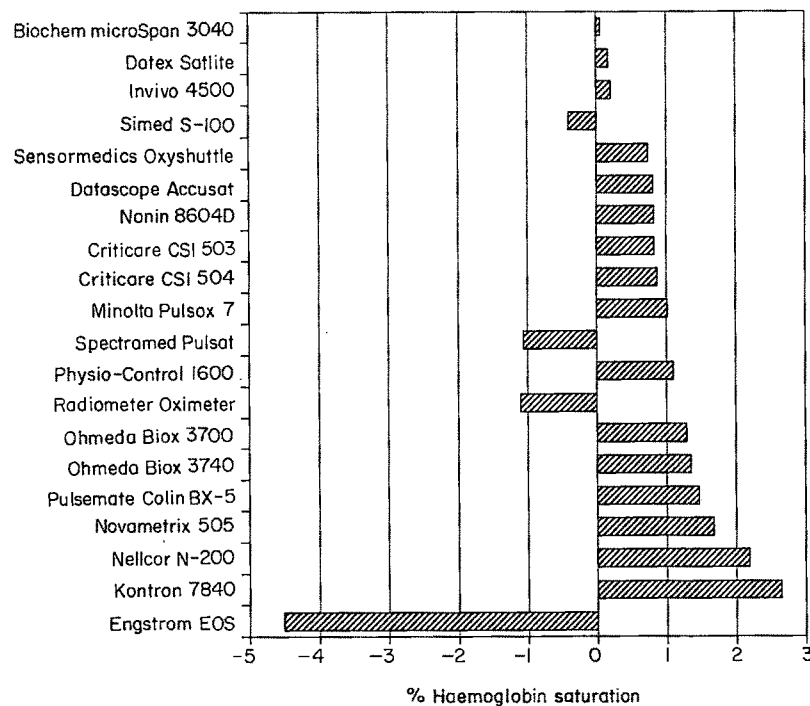


Fig. 2. Pulse oximeter brands ranked by accuracy, or mean of the differences between the pulse oximeter readings and the co-oximeter readings.

cardiac index and temperature at which readings were obtained were 2.4 (litres/minute)/sq m and 26.5°C respectively. Great interindividual variability was characteristic of all variables and equipment studied.

Our results show that under conditions of poor perfusion only two oximeters would be expected to give readings within 4% of our reference co-oximeter 95% of the time. These were the Datex Satlite and Criticare CSI 503. Six

would be expected to give readings within 5%. These were the Invivo 4500, Novamatrix 505, Simed S-100, Nellcor N-200, Physio-Control 1600 and Kontron 7840. Eight would be expected to give readings within 6%. These were the Sensormedics Oxyshuttle, Datascope Accusat, Radiometer OXI, Minolta Pulsox 7, Biochem Microspan 3040, Nonin 8604D, Criticare CSI 504 and PulseMate Colin BX-5. The final four would be expected to give

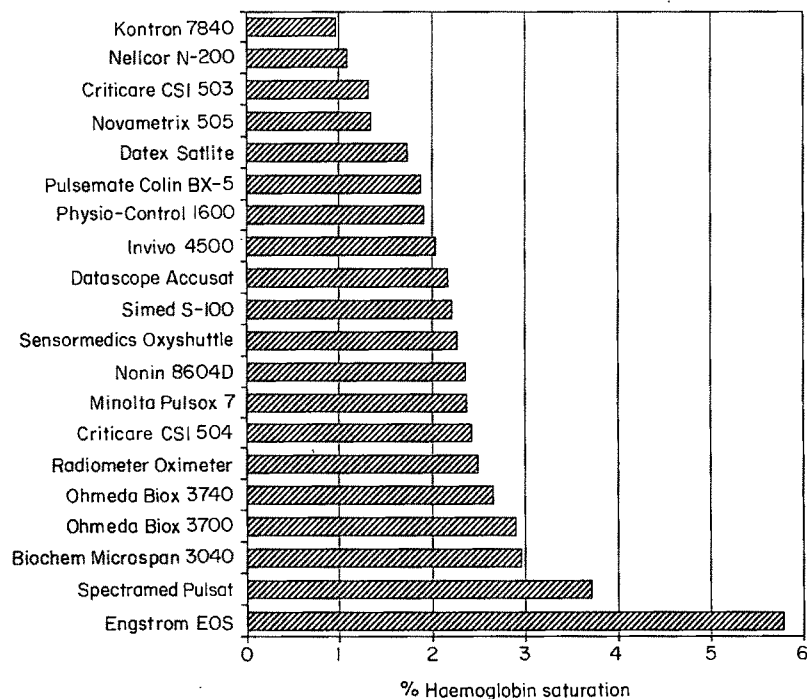


Fig. 3. Pulse oximeter brands ranked by precision, or standard deviation of the differences between the pulse oximeter readings and the co-oximeter readings.



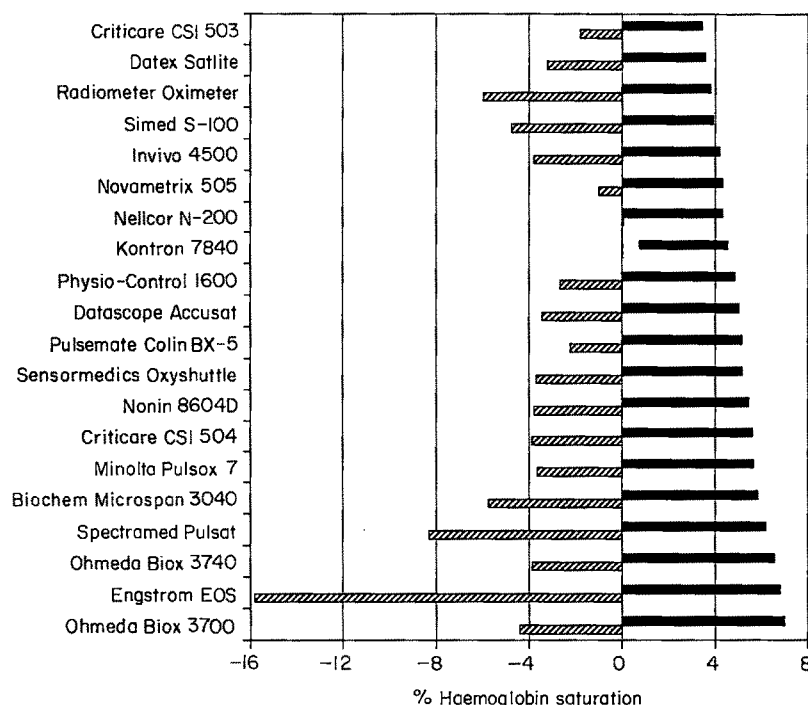


Fig. 4. The 95% limits for each pulse oximeter, with the brands ranked by positive limit.

readings outside these ranges. These were the Ohmeda Biox 3700 and 3740, Spectramed Pulsat and Engstrom Eos. It can be seen that oximeters that performed well tended to overread while some of those that performed poorly, in particular the Engstrom Eos, tended to underread. It may be argued that underreading is safer than overreading since the clinician will react more readily to desaturation, but this does not vindicate the poor overall performance of some of the oximeters studied.

Oximeters gave no reading in 8.7% of measurements. Ten of the 20 oximeters failed to give readings at least 10% of the time.

Whenever a comparative study is performed with a large number of devices there is always a certain amount of pressure exerted upon those performing the study to rank the devices according to some chosen criteria. This is especially so with a device such as the pulse oximeter which is used in acute care clinical situations, cannot be easily calibrated, and if it performs poorly, can lead to patient harm. It is also important to distinguish between assessment of the performance of the device with respect to the manufacturer's specifications, which in this case would be that 95% of values are within 4% of the true value, and performance with respect to clinically acceptable accuracy, which in this study was designated as within 3% of true value. Some clinicians may be unhappy accepting an error of 3%, since a pulse oximeter reading of 93% saturation would mean the patient had a possible arterial oxygen tension of between 59 and 85 mmHg.

The first difficulty is in the choice of criteria to be used for the ranking. One obvious criterion is the accuracy of the device when compared with the best available 'gold standard'. An average difference between the reading given by the pulse oximeter and that given by the co-oximeter is such an example. The pulse oximeters can then be ranked according to the magnitude of that difference (Fig. 2). This raises the question of whether the differences should be

used or whether, as in the case of the pulse oximeter, a device that overreads should be ranked lower than one that 'fails safe' and underreads.

Another criterion is the precision or reproducibility of the measurements. This is determined by the standard deviation of the differences between the pulse oximeter and the co-oximeter and again the pulse oximeters can be ranked in order from smallest to largest standard deviation (Fig. 3).

The pulse oximeters varied quite markedly in their ranking for these two criteria. The Biochem Microspan 3040 ranked number one in terms of accuracy but only number 18 in terms of precision. This means that there were quite wide swings about the true value and while the average difference was small, an individual reading would have a high chance of being inaccurate. On the other hand, the Kontron 7840 ranked number one in terms of precision but only ranked number 19 in terms of accuracy. If an offset of  $-2.65\%$  were applied to the algorithm used in the Kontron 7840 then it would have ranked highest overall of the pulse oximeters tested with respect to accuracy and precision.

The 'gold standard' used in this study was the IL 482 co-oximeter (Lexington, MA, USA). This gives an oxyhaemoglobin saturation (fractional saturation), and thus may tend to favour devices such as the Ohmeda which is calibrated against fractional saturation rather than the Nellcor which is calibrated to give functional saturation. The precision (SD) information would, however, be unaffected; the argument for evaluation of pulse oximeter performance against fractional saturation is discussed in detail in the companion paper.<sup>12</sup>

We also examined the number of readings that each unit gave out of the 40 patients tested in this study. There is again the problem of how these results should be ranked. It can be seen from Figure 1 that some of the units gave readings for all 40 patients but the percentage of these

**Table 4.** Pulse oximeter brands ranked on a combination of accuracy, precision, number of readings within 3% and positive limit.

Oximeter	Accuracy	Precision	±3%	Percent	Positive limit	Combined
Datex Satlite	2	5	2	2	2	13
Criticare CSI 503	8	3	1	1	1	14
Invivo 4500	3	8	5	6	5	27
Novamatrix 505	17	4	3	4	6	34
Simed S-100	4	10	11	12	4	41
Sensormedics Oxyshuttle	5	11	8	6	12	42
Nellcor N-200	18	2	7	10	6	43
Physio-Control 1600	12	7	10	6	9	44
Criticare CSI 504	9	14	3	5	14	45
Datascope Accusat	6	9	14	13	10	52
Radiometer Oximeter	13	15	8	14	3	53
Minolta Pulsox 7	10	13	5	10	15	53
Biochem Microspan 3040	1	18	16	3	16	54
Nonin 8604D	7	12	13	14	13	59
PulseMate Colin BX-5	16	6	11	17	11	61
Kontron 7840	19	1	14	19	8	61
Ohmeda Biox 3740	15	16	16	9	18	74
Spectramed Pulsat	11	19	18	16	17	81
Ohmeda Biox 3700	14	17	18	18	20	87
Engstrom Eos	20	20	20	20	19	99

readings that were of acceptable accuracy (within 3%) varied enormously. How should we rank the results of this part of the study? We attempted to do this by ranking the pulse oximeters in order according to how many readings out of the 40 were within 3% of the co-oximeter (see Fig. 1). We also calculated the percentage of readings, out of the total readings given, that were within 3% of the co-oximeter. The pulse oximeters were then ranked according to this result (see Table 3). This ranking is biased in favour of those units which may not have given as many results as some others, but when they did give a result, it was more likely to be of acceptable accuracy. One unit for example gave readings for all 40 patients but only 27 were within 3% of the co-oximeter (a percentage of 68, ranking of 19). Another unit only gave 28 readings but 26 were within 3% of the co-oximeter (a percentage of 93, ranking of 3).

The fifth method of ranking was to look at the highest positive error that 95% of the units readings would be expected to fall below (see Fig. 4). This makes the assumption that, from a clinical safety point of view, those units which make a lower positive error should be ranked higher than those that make a higher positive error.

We did not perform linear regression analysis and calculation of correlation coefficients for each of the pulse oximeters tested. The issue of which is the appropriate statistical method to interpret the data of methods-comparison studies<sup>13-15</sup> was addressed by Tremper<sup>1</sup> and we agree that calculation of means of difference between the pulse oximeters and the co-oximeter and the standard deviations of these differences gives the most meaningful information.

Other criteria, which include human factors design, alarms and indicators, electrical performance and safety, accuracy in the presence of interference, probe characteristics, operator's manual, quality of construction and ease of servicing, are considered in the Health Devices article by the Emergency Care Research Institute, in which some of the data from the 13 devices studied here and marketed in the USA also appears.<sup>9</sup>

How then should these rankings be combined? Table 4 shows that they were simply added together to give an overall ranking. Others may wish to add a weight to the various individual criteria before they are combined. All the rankings are presented for the five individual criteria (Table 4) for this reason, so that potential purchasers can 'weight' each ranking according to their own preferences.

This study was carried out using oximeters supplied with the then current software. It is likely that most manufacturers have since updated their software, and that the empirically adjusted 'read-out' values will correlate better with a typical group of patients.<sup>12</sup> Nevertheless, this study emphasises that there may be important differences between pulse oximeters, and that further comparative evaluations of pulse oximeters will be necessary.

## References

1. TREMPER, K.K., BARKER S.J. Pulse oximetry. *Anesthesiology* 1989; **70**: 98-108.
2. ALEXANDER CM, TELLER LE, GROSS JB. Principles of pulse oximetry: theoretical and practical considerations. *Anesthesia and Analgesia* 1989; **68**: 368-76.
3. KELLEHER JF. Pulse oximetry. *Journal of Clinical Monitoring* 1989; **5**: 37-62.
4. KIDD JF, VICKERS MD. Pulse oximeters: essential monitors with limitations. *British Journal of Anaesthesia* 1989; **62**: 355-7.
5. MORRIS RW, NAIRN M, TORDA TA. A comparison of fifteen pulse oximeters. Part I. A clinical comparison. Part II A test of performance under conditions of poor perfusion. *Anaesthesia and Intensive Care* 1989; **17**: 62-73.
6. WEBB RK, RALSTON A, CLAYTON D, RUNCIMAN WB. Comparison of pulse oximeters—comment. *Anaesthesia and Intensive Care* 1989; **17**: 238-9.
7. WILKINS CJ, MOORES M, HANNING CD. Comparison of pulse oximeters: effects of vasoconstriction and venous engorgement. *British Journal of Anaesthesia* 1989; **62**: 439-44.
8. KUTTLA KJ, NIINIKOSKI J. Peripheral perfusion after cardiac surgery. *Critical Care Medicine* 1989; **17**: 217-20.
9. EMERGENCY CARE RESEARCH INSTITUTE. Pulse oximeters. *Health Devices* 1989; **18**: 183-230.

10. TREMPER KK, HUFSTEDLER SM, BARKER SJ, ADAMS L, WONG DH, ZACCARI J, BENIK K, LEMONS V. Accuracy of a pulse oximeter in the critically ill adult: effect of temperature and hemodynamics. *Anesthesiology* 1985; **63A**:175.
11. PALVE H, VUORI A. Pulse oximetry during low cardiac output and hypothermia states immediately after open heart surgery. *Critical Care Medicine* 1989; **17**: 66-9.
12. RALSTON AC, WEBB RK, RUNCIMAN WB. Potential errors in pulse oximetry. Pulse oximeter evaluation. *Anaesthesia* 1991; **46**: (in press).
13. ALTMAN DG, BLAND JM. Measurement in medicine: The analysis of method comparison studies. *The Statistician* 1983; **32**: 307-17.
14. BLAND JM, ALTMAN DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet* 1983; 307-10.
15. ALTMAN DG. Statistics and ethics in medical research. Presentation of results. *British Medical Journal* 1980; **281**: 1542-4.



## Regional anaesthesia and cough effectiveness

### A study in patients undergoing Caesarean section

A. W. HARROP-GRIFFITHS, A. RAVALIA, D. A. BROWNE AND P. N. ROBINSON

#### Summary

*We report the results of a study of the effects of spinal and epidural anaesthesia for Caesarean section on commonly used indicators of a patient's ability to cough effectively. Both spinal and epidural anaesthesia, after the achievement of a block adequate for surgery, were associated with statistically significant decreases ( $p < 0.05$ ) in all the respiratory variables recorded: forced vital capacity, forced expiratory volume in one second, peak expiratory flow rate and maximum expiratory pressure. We conclude that although the observed changes are unlikely to impair the normal patient's ability to cough effectively in these circumstances, there may be clinically significant impairment in the presence of an inadvertently high block or in a patient with pre-existing pulmonary disease.*

#### Key words

Anaesthesia; obstetric.

Anaesthetic techniques, regional; epidural, spinal.

Death as a result of the pulmonary aspiration of stomach contents during regional anaesthesia for Caesarean section has been reported.<sup>1</sup> Aspiration during regional anaesthesia may be attributable to loss of protective airway reflexes during periods of compromised consciousness because of drug administration or hypotension. However, it is also conceivable that the extensive motor block of abdominal and intercostal muscles produced during regional anaesthesia for Caesarean section may impair the patient's ability to cough effectively and thereby clear the airways. We report a study of the effects of regional anaesthesia for Caesarean section on commonly used indicators of a patient's ability to cough effectively.

#### Methods

Twenty ASA 1 patients presenting for elective Caesarean section were studied after institutional ethics approval and informed verbal consent. The patients were randomly allocated to one of two equal groups, to receive spinal or epidural anaesthesia. Plain bupivacaine 0.5% was used for both techniques in volumes considered appropriate by the anaesthetist conducting the anaesthetic. Monitoring comprised continuous ECG and finger oxygen saturation and intermittent automated noninvasive arterial blood pressure.

Respiratory function tests comprised forced vital capacity (FVC) and forced expired volume in one second

(FEV<sub>1</sub>) (Vitalograph), peak expiratory flow (PEF) (Wright Peak Flow Meter) and maximum expiratory pressure (PE<sub>max</sub>) (mercury manometer). All patients were trained in the use of the above devices before the start of the study. Respiratory function tests were performed at three times: erect, before anaesthesia (erect/preblock); supine (with 15° left lateral tilt), before anaesthesia (supine/preblock); supine (with 15° left lateral tilt), when the spinal or epidural block was judged adequate for surgery to start (supine/postblock). The recorded value at each time was the highest of three estimations.

Compound sodium lactate solution 1500 ml was given intravenously before establishment of spinal or epidural anaesthesia. Mean arterial pressure was maintained within 30% of pre-operative control values with the use of further compound sodium lactate solution and intravenous ephedrine as appropriate.

Statistical analysis was performed with unpaired Student's *t*-tests, repeated-measures analysis of variance and the Student-Newman-Keuls test.

#### Results

There were no statistically significant differences between the two groups with respect to weight, height, gestational age or block height at supine/postblock respiratory function testing (Table 1). There were no significant differences in heart rate, arterial blood pressure or oxygen saturation

A. W. Harrop-Griffiths, MA, MB, BS, FCAnaes, Locum Consultant, Department of Anaesthesia, St Mary's Hospital, Praed Street, London W2, A. Ravalia, MB, ChB, FFARCSI, Senior Registrar, D. A. Browne, MB, BCh, FFARCSI, Registrar, P. N. Robinson, MB, ChB, FCAnaes, Consultant, Department of Anaesthesia, Edgware General Hospital, Edgware, Middlesex.

Accepted 1 June 1990.

**Table 1.** Mean (SD) weight, height, gestational age, block height and bupivacaine dose in the two groups studied.

	Spinal ( <i>n</i> = 10)	Epidural ( <i>n</i> = 10)
Weight; kg	71.9 (10.6)	73.2 (16.1)
Height; cm	162.6 (5.6)	162.6 (7.3)
Gestational age; weeks	38.7 (1.2)	38.9 (1.2)
Block height; thoracic dermatome	3.8 (1.1)	4.7 (2.5)
Bupivacaine dose; mg	12.1 (2.7)	106.0 (13.1)

**Table 2.** Mean (SD) forced vital capacity (FVC), forced expired volume in one second (FEV<sub>1</sub>), peak expiratory flow rate (PEFR) and maximum expiratory pressure (PE<sub>max</sub>) at three measurements times in the two groups studied.

	Spinal group		
	Erect preblock	Supine preblock	Supine postblock
FVC; litres	3.0 (0.6)	2.8 (0.6)	2.6 (0.5)**
FEV <sub>1</sub> ; litres	2.6 (0.5)	2.4 (0.5)	2.2 (0.5)**
PEF; litres/minute	405 (72)	391 (70)	319 (68)**††
PE <sub>max</sub> ; mmHg	68 (27)	60 (28)	46 (17)**†
	Epidural group		
	Erect preblock	Supine preblock	Supine postblock
FVC; litres	2.9 (0.5)	2.7 (0.4)**	2.5 (0.5)**†
FEV <sub>1</sub> ; litres	2.5 (0.5)	2.2 (0.5)*	2.0 (0.5)**
PEF; litres/minute	371 (77)	344 (64)*	319 (62)**
PE <sub>max</sub> ; mmHg	79 (23)	72 (23)	59 (22)**††

Statistical differences: \* compared with erect/preblock ( $p < 0.05$ ); \*\* compared with erect/preblock ( $p < 0.01$ ); † compared with supine/preblock ( $p < 0.05$ ); †† compared with supine/preblock ( $p < 0.01$ ).

within or between the two groups during the conduct of the study.

Table 2 shows the mean values of FVC, FEV<sub>1</sub>, PEF and PE<sub>max</sub> at the three measurement times. There were decreases of between 3 and 12% in all mean values at the supine/preblock measurement when compared with erect/preblock controls. There were further decreases of between 7 and 23% after administration of spinal and epidural anaesthesia. The greatest overall decreases were seen in PE<sub>max</sub>, which were 32% and 25% respectively for the spinal and epidural groups when compared with erect/preblock controls. There were no significant differences between the two groups at any of the three measurement times.

### Discussion

There is no uniform agreement on which respiratory function test is the best indicator of a patient's ability to cough. The performance of a cough under normal circumstances depends upon the ability to inspire deeply, close the glottis and to increase intrapulmonary pressure.<sup>2</sup> It is likely that the abdominal muscles are the most important muscles in the production of effective cough since they are primarily responsible for increasing intra-abdominal pressure, and thereby intrapulmonary pressure, before an expulsive effort.<sup>3</sup> The flow profile of human cough has two phases.<sup>4</sup> An initial phase that lasts 30 to 50 mseconds, characterised by gas flows of up to 750 litres/minute and a second more prolonged phase with flows in the order of 250 litres/minute.

A study reported by King, Brock and Lundell<sup>4</sup> made use of a device to simulate the gas flow profile of a human cough and relate changes in the gas flow profile to the clearance of mucus from a simulated trachea. Their results indicate that the initial phase of cough, likely to be related to intrapulmonary pressure, is the phase that is more effective in clearing mucus from the trachea; the second phase, more related to airways resistance, is not as effective. PEF is known to be an indicator of changes in airways resistance and can therefore be presumed to give an indication of gas flow in the second phase rather than the first. It therefore appears that although FVC, PEF and FEV<sub>1</sub> bear some proportional relation to the ability to cough, it is reasonable to assert that PE<sub>max</sub> may be a more sensitive indicator of impairment of cough function. Previous studies of respiratory function during regional anaesthesia<sup>5-11</sup> have consistently reported decreases in FVC and PEF, though the changes have not always achieved statistical significance. A study reported by Moir<sup>12</sup> on patients who undergo abdominal surgery under epidural anaesthesia showed small decreases in FVC and PEF and led him to conclude that it was 'unlikely that the ability to cough is much impaired'. Egbert and others<sup>13</sup> studied healthy men undergoing inguinal herniorrhaphy under spinal anaesthesia and, demonstrating substantial decrease in PE<sub>max</sub>, concluded that their ability to cough was significantly impaired. The only previous study<sup>14</sup> of respiratory function during regional anaesthesia for Caesarean section that we are aware of restricted itself to measurement of PEF. The decreases in PEF observed after regional anaes-

thesia were not considered sufficient to signify an impairment of cough function.

Our results show that there are statistically significant decreases in FVC, FEV<sub>1</sub>, PEF and PE<sub>max</sub> after regional anaesthesia when compared with values obtained in the erect position. The greatest decreases were seen in PE<sub>max</sub>. We conclude that although these effects are unlikely seriously to affect the normal patient's ability to cough when undergoing Caesarean section under regional anaesthesia, there may be a danger in this respect after inadvertently high blocks or in patients who have pre-existing pulmonary disease. The fact that a reported death after aspiration during epidural anaesthesia for Caesarean section occurred in a patient with pre-existing pulmonary disease<sup>1</sup> may lend credence to this hypothesis.

### References

1. Department of Health and Social Security. Report on confidential enquiries into maternal deaths in England and Wales 1976-1978. London: HMSO, 1982: 80.
2. BUCHER K. Pathophysiology and pharmacology of cough. *Pharmacological Reviews* 1958; **10**: 43-58.
3. CAMPBELL EJM, AGOSTINI E, NEWSOM DAVIS J. *The respiratory muscles: mechanics and neural control*. London: Lloyd-Luke, 1970.
4. KING M, BROCK G, LUNDELL C. Clearance of mucus by simulated cough. *Journal of Applied Physiology* 1985; **58**: 1776-82.
5. WAHBA WM, CRAIG DB, DON HF, BECKLAKE MR. The cardio-respiratory effects of thoracic epidural anaesthesia. *Canadian Anaesthetists' Society Journal* 1972; **19**: 8-19.
6. SUNDBERG A, WATTWIL M, ARVILL A. Respiratory effects of high thoracic epidural anaesthesia. *Acta Anaesthesiologica Scandinavica* 1986; **30**: 215-7.
7. SJOGREN S, WRIGHT B. Respiratory changes during continuous epidural blockade. *Acta Anaesthesiologica Scandinavica* (Suppl.) 1972; **46**: 27-49.
8. TAKASAKI M, TAKAHASHI T. Respiratory function during cervical and thoracic extradural analgesia in patients with normal lungs. *British Journal of Anaesthesia* 1980; **52**: 1271-75.
9. DRUMMOND GB, LITTLEWOOD DG. Respiratory effects of extradural analgesia after lower abdominal surgery. *British Journal of Anaesthesia* 1977; **49**: 999-1004.
10. FREUND FG, BONICA JJ, WARD RJ, AKAMATSU TJ, KENNEDY WF. Ventilatory reserve and level of motor block during high spinal and epidural anesthesia. *Anesthesiology* 1967; **28**: 834-7.
11. MCCARTHY GS. The effect of thoracic extradural analgesia on pulmonary gas distribution, functional residual capacity and airway closure. *British Journal of Anaesthesia* 1976; **48**: 243-7.
12. MOIR DD. Ventilatory function during epidural analgesia. *British Journal of Anaesthesia* 1963; **35**: 3-7.
13. EGBERT LD, TAMERSOY K, DEAS TC. Pulmonary function during spinal anesthesia: the mechanism of cough depression. *Anesthesiology* 1961; **22**: 882-5.
14. GAMIL M. Serial peak expiratory flow rates in mothers during Caesarean section under extradural anaesthesia. *British Journal of Anaesthesia* 1989; **62**: 415-18.



## Effect of glyceryl trinitrate and ice on dilation of hand veins

S. WILLIAMS AND J. F. HECKER

### Summary

*Application of glyceryl trinitrate to a finger produced significant dilation of superficial hand veins. Gently rubbing ice over these veins for one minute slightly reduced this dilation, whereas rubbing ice over veins without glyceryl trinitrate caused significant venoconstriction. These results indicate that application of glyceryl trinitrate followed by local cooling may provide a simple and painless means of assisting venepuncture.*

### Keywords

*Veins; venepuncture.*

*Pharmacology; glyceryl trinitrate.*

Venepuncture is difficult in some patients because their superficial hand and arm veins often remain small after application of tourniquets, and resort has to be made to 'aids' such as warming the limb, tapping or rubbing the skin over a selected vein or having the patient repeatedly clench and unclench a hand to cause a vein to dilate. We have previously shown that hand and arm veins dilate and that cannulation is made easier if a small amount of glyceryl trinitrate (GTN) is applied transcutaneously a few minutes before attempting cannulation. Several other papers, since we described this technique,<sup>1</sup> have confirmed that GTN dilates veins intended for venepuncture.<sup>2–6</sup> Most patients are apprehensive of venepuncture because they anticipate pain. Cooling of skin provides anaesthesia, but venepuncture is made more difficult because veins beneath the cooled skin constrict. This paper presents the results of an experiment designed to determine whether prior local application of GTN reduces the venoconstrictive response to local cooling.

### Methods

The experiment was given Ethics Committee approval, and 19 volunteer patients (12 males and seven females) gave informed consent. They stood during measurements but remained in bed at other times. Each was allocated a pair of Transiderm-Nitro 5 systems (CIBA) marked left and right. One of each pair was active and the other was a placebo allocated according to a set of random numbers.

One vein on each hand was selected as suitable for venepuncture and was marked. Each hand was hung by the side for one minute before the diameter of the marked vein was measured to the nearest 0.2 mm with a set of callipers. Blood pressure cuffs around the arms were then inflated to diastolic blood pressure and the measurements were repeated after a further minute. The 'left' and 'right' Transiderm-Nitro systems were placed around the bases of the second fingers of the respective hands and secured with a short length of tape after these measurements were made. Measurements were repeated one hour later during which time patients had returned to their beds, first with hands hanging for one minute and then after cuffs were applied at diastolic pressure for one minute. Finally, a block of ice was gently moved over the marked sites for one minute and measurements were again made with the hands hanging and then with the cuffs.

Eight replicate measurements were made at 2-minute intervals on a marked vein on a hand hung down. The mean size was 5.83 (SD 0.11) mm.

Statistical analysis was by analysis of variance on the four main factors (sex, time, GTN and dilation method (cuff)) and their interactions using the 2 V (repeated measures) program of the BMDP statistical package.<sup>7</sup>

### Results

The mean vein diameters are shown in Table 1 and results from the analysis of variance in Table 2. The difference was

S. Williams,\* MB, ChB, FRCS, J. F. Hecker,† PhD, BVSc, Department of Surgery Royal Postgraduate Medical School, DuCane Road, London.

\*Present addresses: Lily Research Centre Ltd, Erl Wood Manor, Windlesham, Surrey. †Department of Physiology, The University of New England, Armidale, NSW, Australia 2351.

Correspondence should be addressed to Dr J. F. Hecker please.

Accepted 11 June 1990.

**Table 1.** Size of marked veins on hands with active or placebo Transiderm-Nitro devices. Size was measured after letting hands hang and after applying a cuff at diastolic blood pressure. Ice measurements were made after gently moving ice over the vein for 1 minute. Values are expressed as mean (SEM).

Measurement	Active (mm)	Placebo (mm)
<i>Males</i>		
Dependent without cuff		
Initial	3.70 (0.24)	3.78 (0.24)
After 1 hour	5.00 (0.37)	3.93 (0.22)
After 1 hour, ice	4.75 (0.29)	3.02 (0.25)
Dependent with cuff		
Initial	4.88 (0.25)	4.62 (0.28)
After 1 hour	6.10 (0.32)	4.87 (0.24)
After 1 hour, ice	5.80 (0.33)	4.25 (0.23)
<i>Females</i>		
Dependent without cuff		
Initial	3.14 (0.22)	3.03 (0.20)
After 1 hour	4.34 (0.27)	2.23 (0.22)
After 1 hour, ice	4.06 (0.24)	2.69 (0.15)
Dependent with cuff		
Initial	3.83 (0.22)	3.81 (0.24)
After 1 hour	5.03 (0.25)	3.97 (0.20)
After 1 hour, ice	4.97 (0.27)	3.37 (0.13)

highly significant as regards dilation method, GTN and time factors but although veins were smaller for females this difference was not significant. The only significant interactions were GTN and time and dilation method and time although the interaction between all four factors almost reached the  $p < 0.05$  level. Cuffs at diastolic pressure caused diameters to increase an overall average of 0.90 mm more than when the hands were just hanging, with a significant difference for each set of measurements. The mean increase in diameter (averaged for sex, hand hanging and cuff measurements) one hour after application of the GTN was 0.2 mm for placebo devices, but was not significant, whereas the 0.96 mm increase for active devices was significant. The effect of rubbing ice was a significant net decrease in size of 0.50 mm compared with initial values in hands with control devices and a significant net increase of 0.51 mm compared with initial values in hands with active devices.

### Discussion

The veins were measured at two dilating pressures. Hands hung were about 70 cm lower than the clavicle and would therefore have had a dilating pressure when filled of about 50 mmHg. Diastolic blood pressures were in the order 80 mmHg (range 70–90 mmHg) and, as the hands would have been about 40 cm below the cuff, the hand vein pressure would have been approximately 110 mmHg when the cuff was inflated. The overall increase in diameter produced by the GTN was approximately 2.4 mm with the cuff and 1.4 mm without the cuff which suggests that the relation between dilation and dilating pressure was not linear. The significant interaction between cuff and sex was apparently due to the cuff producing greater dilation compared with hanging in males.

We had a choice of alternative forms of GTN available, such as transcutaneous creams and sublingual sprays but we selected the Transiderm-Nitro systems since they would provide the most reproducible dose of GTN. About 8% of the drug (0.4 mg for a 5 mg system) is dissolved in the

**Table 2.** Data from the analysis of variance for the variable and the significant interactions.

Source	Degrees of freedom	Mean square	F	p
GTN	1	4595.60	21.04	0.0003†
Cuff	1	4330.48	351.83	0.0000§
Time	2	911.21	43.59	0.0000§
Sex	1	3120.74	3.01	0.101 (ns)
Cuff × sex	1	124.96	10.15	0.0054†
GTN × time	2	1028.62	16.94	0.0000§
GTN × cuff × time × sex	2	21.28	3.25	0.051 (ns)

GTN: active or placebo systems; cuff: hands dependent with or without cuff; time: initial, after 1 hour or after ice.

(ns) not significant, † $p < 0.01$ , ‡ $p < 0.001$ , § $p < 0.0001$ .

adhesive layer and is available for rapid absorption.<sup>8</sup> One hour was allowed for absorption (another 0.2 mg might be absorbed in this hour) to eliminate any effect from variations in skin permeability, although veins usually dilate within a few minutes after transcutaneous application of GTN. A possible counter-current exchange mechanism between deep arteries and veins in the arm<sup>9</sup> could enhance the effect.

The mean increase in size from GTN plus cuffs was 62%. Lohmann *et al.*<sup>2</sup> reported increases in vein size of 39% while another study<sup>10</sup> indicates an increase of 120%, although their net increase was probably less since control veins also increased by 30% between measurements. Few details were given in these papers of how the measurements were made.

Some subjects complained of discomfort from cold after ice was rubbed for 60 seconds. This period was chosen to maximise the constrictive effect of cooling but we believe that about 30 seconds rubbing might be sufficient to produce a reasonable degree of analgesia if venepuncture is performed immediately the ice is removed. Usually, it would be briefly explained to the patient why ice was being rubbed on and therefore an analgesic 'placebo effect' could be expected with this procedure. A longer period of cold is usually necessary with veins of young children to provide painless venepuncture (G.C. Fisk, personal communication).

Two other techniques are sometimes used to reduce the discomfort of venepuncture. Local anaesthetic creams such as lignocaine-prilocaine creams (EMLA), although they are expensive, may be applied at the proposed venepuncture site and covered with occlusive dressings.<sup>11</sup> In addition, a short time before cannulation with large catheters and cannulae, lignocaine is sometimes injected adjacent to a selected vein with a fine needle,<sup>12</sup> but most patients dislike any needle, including the small sizes used for subdermal injection.

The main advantage of this technique compared with the use of local anaesthetic drugs is that it produces venodilation as well as a degree of analgesia. Another advantage is that all veins in the lower arm will be dilated by the local GTN, and ice could quickly be applied to another vein if the first venepuncture was unsuccessful. There is also no cream or occlusive dressing to be removed from the vein and the local anatomy is not disturbed by an injection.

Transiderm-Nitro systems have been shown to increase the survival of peripheral intravenous infusions by reducing the incidence of phlebitis and extravasation.<sup>13,14</sup> The systems are, however, relatively expensive for routine hos-

pital use as an aid to venepuncture, but a tube of GTN cream would contain sufficient for hundreds of applications. Use of a GTN system would be indicated if it were to be used as an aid for cannulation for an infusion, because there would be the bonus of increased infusion survival if the system was left in place.

### Acknowledgments

We thank Dr G.C. Fisk who has used this technique successfully with children for several years for bringing it to our attention.

### References

- HECKER JF, LEWIS GBH, STANLEY H. Nitroglycerine ointment as an aid to venepuncture. *Lancet* 1983; **1**: 332-3.
- LOHMANN M, MOLLER P, BRYNITZ S, BJERRUM OW. Nitroglycerine ointment as an aid to venepuncture. *Lancet* 1984; **1**: 1416-7.
- MANIFOLD IH, COLE R, MACKINTOSH M, SMITH W, CHAMPION AE, HANCOCK BW. A double blind trial of nitroglycerine vasodilator ointment in patients undergoing cytotoxic chemotherapy. *European Journal of Surgical Oncology* 1986; **12**: 67-8.
- MOORE PL. Nitroglycerin improves venous cannulation. *Anesthesiology* 1986; **64**: 533.
- PARAKH SC, PATWARI A. Experience with Nitrobid (2% nitroglycerine) ointment as an aid to venepuncture. *British Journal of Anaesthesia* 1986; **58**: 822.
- VAKSMANN G, REY C, BREVIERE G-M, SMADJA D, DUPUIS C. Nitroglycerine ointment as aid to venous cannulation in children. *Journal of Pediatrics* 1987; **111**: 89-91.
- BMDP Statistical Software, 1964 Westwood Blvd., Suite 202, Los Angeles, Ca 90025, USA.
- CIBA Ltd, Promotional literature.
- HECKER JF. Is there counter-current exchange of drugs in limbs after percutaneous absorption? *Clinical Pharmacokinetics* 1989; **17**: 141-4.
- ROBERGE RJ, KELLY M, EVANS TC, HOBBS E, SAYRE M, COTTINGTON E. Facilitated intravenous access through local application of nitroglycerin ointment. *Annals of Emergency Medicine* 1987; **16**: 546-9.
- MCKAY W, MORRIS R, MUSHLIN P. Sodium bicarbonate attenuates pain on skin infiltration with lidocaine, with or without epinephrine. *Anesthesia and Analgesia* 1987; **66**: 572-4.
- MAUNUKSELA E-L, KORPELA R. Double-blind evaluation of a lignocaine-prilocaine cream (EMLA) in children: Effect on the pain associated with venous cannulation. *British Journal of Anaesthesia* 1986; **58**: 1242-5.
- KHAWAJA HT, CAMPBELL MJ, WEAVER PC. Effect of transdermal glyceryl trinitrate on the survival of peripheral intravenous infusions: a double-blind prospective clinical study. *British Journal of Surgery* 1988; **75**: 1212-5.
- WRIGHT A, HECKER JF, LEWIS GBH. Use of transdermal glyceryl trinitrate to reduce failure of intravenous infusion due to phlebitis and extravasation. *Lancet* 1985; **2**: 1148-50.



## Influence of temperature of bupivacaine on spread of spinal analgesia

T. CALLESEN, I. JARNVIG, B. THAGE, T. KRANTZ AND C. CHRISTIANSEN

### Summary

*A prospective, randomised study was performed to investigate the influence of temperature on sensory blockade in spinal anaesthesia. Three ml of plain bupivacaine 0.5% were injected intrathecally at either 4°C, room temperature, or 37°C. There were 10 patients in each group, who were kept sitting for 2 minutes after injection. The maximum level of sensory blockade was significantly higher ( $p < 0.01$ ) in the group who received the solution adjusted to 37°C, and variability of level was smaller ( $p < 0.05$ ). Time to two-segment regression was shorter in the 37°C group than in the 4°C group ( $p < 0.05$ ). Hypotension required administration of ephedrine more often in the 37°C group ( $p < 0.05$ ). It is concluded that the use of plain bupivacaine 0.5% adjusted to 37°C results in a higher and more predictable sensory blockade.*

### Key words

*Anaesthetics, local; bupivacaine.  
Anaesthetic technique, regional; spinal.  
Temperature.*

Many factors are claimed to affect the spread of local anaesthetics used for spinal anaesthesia.<sup>1</sup> Among these is baricity, defined as the ratio between the densities of the local anaesthetic solution and the cerebrospinal fluid (CSF). The temperatures of the solutions are essential to the calculation of baricity, since density varies inversely with temperature. The mean density of the CSF is 1.0001 g/cm<sup>3</sup> at 37°C,<sup>2</sup> and the density of plain bupivacaine 0.5% varies from 1.003 g/cm<sup>3</sup> at 4°C to 0.997 g/cm<sup>3</sup> at 37°C (manufacturer's information). Cold, plain bupivacaine might therefore act as a hyperbaric solution until equilibration to body temperature has occurred, which has been estimated to take 1 to 2 minutes.<sup>3</sup>

The present study was undertaken to investigate the influence of the temperature of the local anaesthetic solution on the spread of spinal anaesthesia using 0.5% plain bupivacaine.

### Methods

Thirty males, aged 50 to 80 years and 165 to 185 cm in height, scheduled for urological or lower extremity surgery were studied after Ethics Committee approval and informed consent. The patients were randomly allocated to one of three groups to receive 0.5% plain bupivacaine

adjusted to either 4°C (group 1), room temperature (group 2) or 37°C (group 3). Both patients and observers were blind to the allocation.

One litre of isotonic saline was infused at the arrival in the operating theatre, after premedication with morphine. Baseline arterial blood pressure and heart rate were recorded.

Lumbar puncture was performed in the sitting position in the midline at the L<sub>3</sub>–L<sub>4</sub> interspace using a 26-gauge spinal needle. Three ml of the plain bupivacaine were injected during 15 seconds using a syringe and a needle equilibrated to the same temperature as the bupivacaine solution. Heart rate and arterial blood pressure were recorded after 2 minutes, and the patients placed supine. Analgesia to pinprick, tested bilaterally in the midclavicular lines, heart rate and blood pressure were recorded 5, 10, 15, 20, 25, 30, 40, 50, and 60 minutes after injection. These variables were then recorded every 30 minutes until a four-segment regression in the height of the block had occurred, or at least for 3.5 hours.

Motor blockade was assessed at 30 minutes as described by Bromage;<sup>4</sup> 0 represented no muscle weakness and 3 complete paralysis of the lower extremities. The Kruskal–Wallis test and the squared rank test for inequality of variance for several samples were used for statistical

T. Callesen, MD, I. Jarnvig, MD, B. Thage, MD, Registrars, T. Krantz, MD, Consultant, C. Christiansen, MD, Consultant, Chief Physician, Department of Anaesthesiology and Intensive Care, Frederiksberg Hospital, Ndr. Fasanvej 57, DK 2000 Copenhagen F, Denmark.

Correspondence should be addressed to Dr T. Callesen, Skytteengen 14, DK 3450 Allerød, Denmark.

Accepted 25 June 1990.

**Table 1.** Age, height, and weight of the three groups. Values are expressed as mean (SD).

	Age; years	Height; cm	Weight; kg
Group 1; 4°C	73.0 (4.9)	176 (4.2)	73.6 (8.7)
Group 2; room temperature	70.8 (7.4)	173 (5.2)	74.9 (8.4)
Group 3; 37°C	68.7 (8.0)	176 (4.7)	78.3 (14.7)

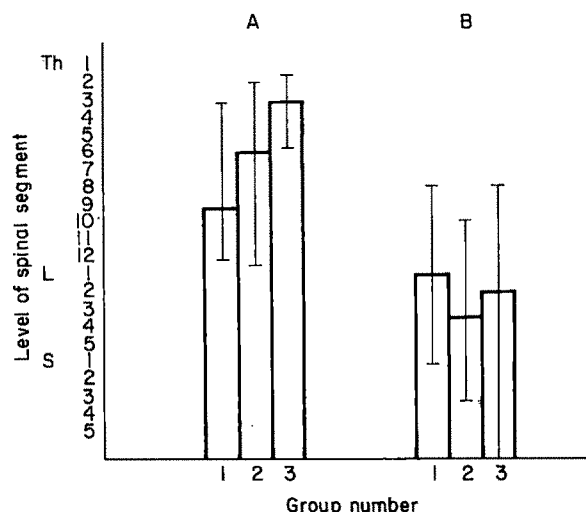
**Table 2.** Maximum level of sensory blockade and time for two-segment regression. Values are expressed as median (range).

	Maximum height of block; spinal level	Time for 2 segment regression; hours
Group 1; 4°C	Th 9 (Th <sub>12</sub> -Th <sub>3</sub> )	2.25 (1.5-4.5)
Group 2; room	Th 6 (L <sub>1</sub> /Th <sub>12</sub> -Th <sub>2</sub> )	2.0 (1.5-2.5)
Group 3; 37°C	Th 3 (Th <sub>5</sub> /Th <sub>6</sub> -Th <sub>1</sub> /Th <sub>2</sub> )	1.5 (1.0-3.0)

analysis of the level of sensory blockade, time to onset/regression, and changes in blood pressure. For overall significance the Mann-Whitney *U*-test and the squared rank test for two samples were used. Differences in frequencies were tested with the Chi-squared test. A probability of less than 5% was considered significant.

### Results

Data are presented as median and range, when not stated otherwise. The three groups did not differ with respect to age, height and weight (Table 1). In group 3, the height of the block was significantly higher than in groups 1 and 2 ( $p < 0.01$ ,  $p_{1-3} < 0.01$ ,  $p_{2-3} < 0.01$ ); the difference between groups 1 and 2 was not significant (Table 2 and Fig. 1). The range in the maximum levels were 9, 10.5 and 4 segments in groups 1, 2 and 3 respectively and also showed significant differences ( $p < 0.05$ ); group 3 had a significantly smaller range than the two other groups ( $p_{1-3} < 0.05$ ,  $p_{2-3} < 0.05$ ) (Table 2 and Fig. 1). The times (minutes) needed for maximum spread were 12.5 (5-60) in group 1, 25 (10-60) in group 2 and 17.5 (10-40) in group 3. These differences were not statistically significant.



**Fig. 1.** Level of sensory blockade. A, maximum level; B, level 3.5 hours after injection of bupivacaine. Group 1, 4°C; group 2, room temperature; group 3, 37°C. Values are expressed as median and range.

The time to two-segment regression of sensory blockade was significantly longer in group 1 than in group 3 ( $p < 0.05$ ,  $p_{1-3} < 0.05$ ). No such difference could be detected between groups 1 and 2 or groups 2 and 3.

The levels of sensory blockade after 3.5 hours were L<sub>1</sub> (S<sub>1</sub>-Th<sub>8</sub>) in group 1, L<sub>3</sub>-L<sub>4</sub> (S<sub>3</sub>-Th<sub>10</sub>) in group 2, and L<sub>2</sub> (no analgesia-Th<sub>8</sub>) in group 3. These differences were not statistically significant. All 30 patients had a complete motor blockade after 30 minutes.

The decrease in arterial blood pressure necessitated intravenous administration of ephedrine in three patients in group 1, two in group 2 and all patients in group 3. This difference was statistically significant ( $p < 0.05$ ). One patient in group 3 was given atropine because of severe bradycardia. Small doses of opioids and tranquillisers were administered to two, three, and four patients in groups 1, 2 and 3, respectively.

### Discussion

A major problem in spinal anaesthesia is the unpredictable spread of the local anaesthetic solution.<sup>5</sup> Different physical characteristics of the local anaesthetics have been investigated in order to overcome this problem, in particular baricity, dosage and volume of the injectate.<sup>5-7</sup>

Stienstra *et al.*<sup>8,9</sup> investigated the effect of temperature of the local anaesthetic solution on spread of spinal anaesthesia in the sitting position. They found a higher and less variable block using 3 ml 0.5% plain bupivacaine injected at 37°C compared to 4°C. They compared the same solution at 20°C and 37°C in their second study and found that sensory blockade was higher, variability was less and duration of the blockade above specified thoracic dermatomes was longer in the 37°C group.<sup>9</sup> Onset of sensory blockade and time needed for two-segment regression did not differ significantly.

Our findings are mainly in accordance with those of these workers. In addition we have demonstrated an equally high variability in maximum level of sensory blockade in the 4°C and the room temperature groups. No significant difference in sensory level was found between these two groups.

The different densities of bupivacaine 0.5% at 4°C, 20°C, and 37°C and hence the correspondingly different states of baricity (hyper-, iso- and hypobaricity), seems the most reasonable explanation of these findings. The high variability in level of sensory blockade at the lower temperatures

might be explained by the fact that when injected at 4°C or at room temperature the injectate undergoes a change in baricity from hyper-/isobaricity to hypobaricity in the first, seemingly very important, 1 to 2 minutes after the injection.<sup>3</sup> The baricity of the bupivacaine solution is only stable when injected at 37°C.

We also observed a significantly shorter time to regression of two segments in the 37°C group than in the 4°C group. This might be explained by the more widespread distribution and hence the lower concentration of the local anaesthetic in the former 37°C group. There were also no differences in the present study in the time to maximum level of sensory blockade; this confirms the findings of Stienstra *et al.*<sup>8</sup>

Administration of ephedrine was left to the judgment of the observer. It was required significantly more often in those who received bupivacaine at 37°C, where the sensory and probably also the sympathetic blockade was highest.

In conclusion, the use of 3 ml 0.5% plain bupivacaine for spinal anaesthesia in the sitting patient led to a higher and more predictable sensory block when a 37°C solution was compared to both 4°C and room temperature solutions. No significant differences in level or predictability were seen when the 4°C and the room temperature solutions were compared.

## References

1. GREENE NM. Distribution of local anesthetic solutions within the subarachnoid space. *Anesthesia and Analgesia* 1985; **64**: 715–30.
2. DAVIS H, KING WR. Densities of cerebrospinal fluid of human beings. *Anesthesiology* 1954; **15**: 666–72.
3. ERNST AE. *In-vitro* changes of osmolality and density of spinal anesthetic solutions. *Anesthesiology* 1968; **29**: 104–9.
4. BROMAGE PR. A comparison of the hydrochloride and carbon dioxide salts of lidocaine and prilocaine in epidural analgesia. *Acta Anaesthesiologica Scandinavica* 1965; **16** (Suppl.): 55–69.
5. LOGAN MR, MCCLURE JH, WILDSMITH JAW. Plain bupivacaine: an unpredictable spinal anaesthetic agent. *British Journal of Anaesthesia* 1986; **58**: 292–6.
6. MÖLLER IW, FERNANDES A, EDSTRÖM HH. Subarachnoid anaesthesia with 0.5% bupivacaine: effects of density. *British Journal of Anaesthesia* 1984; **56**: 1191–4.
7. SHESKEY MC, ROCCO AG, BIZZARRI-SCHMID M, FRANCIS DM, EDSTRÖM H, COVINO BG. A dose-response study of bupivacaine for spinal anaesthesia. *Anesthesia and Analgesia* 1983; **62**: 931–5.
8. STIENSTRA R, VAN POORTEN JF. The temperature of bupivacaine 0.5% affects the sensory level of spinal anaesthesia. *Anesthesia and Analgesia* 1988; **67**: 272–6.
9. STIENSTRA R, GIELEN M, VAN POORTEN JF, KROON JW. Spinal anaesthesia with plain bupivacaine 0.5%: regression of sensory and motor blockade with different temperatures of the anesthetic solution. *Anesthesia and Analgesia* 1989; **69**: 593–7.

## Propofol for induction and maintenance of anaesthesia at Caesarean section

### A comparison with thiopentone/enflurane

G. YAU, T. GIN, M. C. EWART, C. F. KOTUR, R. K. W. LEUNG AND T. E. OH

#### Summary

*A propofol infusion regimen and a standard general anaesthetic were compared in 40 Chinese women undergoing elective Caesarean section. Twenty patients received propofol 2 mg/kg for induction of anaesthesia followed by propofol 6 mg/kg/hour, while 20 patients received thiopentone 4 mg/kg with enflurane 1% for maintenance of anaesthesia. All patients were given atracurium and their lungs ventilated with nitrous oxide 50% in oxygen until delivery of the neonate. The hypertensive response after intubation was of shorter duration in the propofol group compared with the thiopentone group. Induction to delivery times ranged from 5 to 14 minutes and neonates from both groups had similar and satisfactory Apgar scores, Neurologic and Adaptive Capacity Scores and umbilical cord blood gas analysis. However, a prolonged propofol infusion time before delivery may cause lower Neurologic and Adaptive Capacity Scores. There were no differences in maternal recovery times or psychomotor performance.*

#### Key words

*Anaesthesia; obstetric.*

*Anaesthetics, intravenous; propofol.*

*Anaesthetic techniques; intravenous.*

Recovery from propofol infusions is rapid<sup>1–4</sup> and several groups have evaluated this technique for anaesthesia during elective Caesarean section.<sup>5,6</sup> It was postulated that, as well as rapid maternal recovery, the neonate may also recover quickly after delivery. However, placental transfer of propofol is rapid<sup>7</sup> and infusion techniques deliver a large dose of drug to the fetus, especially if induction to delivery times are prolonged.

A preliminary study with 10 patients per group found that a propofol infusion regimen with 100% oxygen was unsatisfactory, partly because analgesia was not provided before delivery and a propofol infusion with nitrous oxide was more promising.<sup>6</sup> In this study, we compared a propofol infusion plus nitrous oxide with a thiopentone, enflurane and nitrous oxide general anaesthetic.

#### Method

The protocol was approved by the Research Ethics Committee of the Chinese University Faculty of Medicine and informed consent obtained from all patients. Forty ASA I Chinese women undergoing elective Caesarean

section for a normal, singleton pregnancy were randomly assigned to a thiopentone or propofol group.

Oral ranitidine 150 mg was given the night before, and again on the morning of surgery. The patients were transported to the operating theatre in the lateral position and a 15° left lateral tilt was maintained on the operating table. Oral sodium citrate 0.3 M 30 ml was given 15 minutes before induction of anaesthesia. Routine monitoring included pulse oximetry, end-tidal carbon dioxide concentration (Normocap, Datex), electrocardiogram and arterial blood pressure (Dinamap 1846SXP, Critikon Inc). The lungs of each patient were pre-oxygenated for 3 minutes. Anaesthesia was induced, after a baseline arterial blood pressure was recorded, with either thiopentone 4 mg/kg or propofol 2 mg/kg over 20 seconds followed by suxamethonium 1.5 mg/kg. Laryngoscopy followed the next Dinamap recording at one minute and tracheal intubation was accomplished before the second minute recordings. Cricoid pressure was applied from the beginning of the induction sequence until the trachea was intubated and the cuff tested for leaks. The lungs of both groups were ventilated with nitrous oxide 50% in oxygen. In the propofol group, an

G. Yau, MB, BS, FFARCS, T. Gin, MB, ChB, BSc, FFARCS, FFARCS, Lecturers, C.F. Kotur, MB, BS, DA, FFARCS, Honorary Clinical Lecturer, T.E. Oh, MB, BS, FFARCS, FFARCS, Professor, Department of Anaesthesia and Intensive Care, R.K.W. Leung, MB, BS, MRCP, Department of Paediatrics, Prince of Wales Hospital, Chinese University of Hong Kong, Shatin, Hong Kong, M.C. Ewart, MB, BS, FFARCS, Senior Registrar, Hammersmith Hospital, Du Cane Road, London W12 0NN.

Accepted 25 June 1990.



infusion of propofol 6 mg/kg/hour was started immediately after the induction dose while the thiopentone group received enflurane 1% when controlled ventilation was started. Neuromuscular block was continued with atracurium 0.5 mg/kg and ventilation was controlled to maintain end-tidal carbon dioxide concentrations of 4.0–4.5%. A 500-ml infusion of compound sodium lactate solution was given over the first 10 minutes. Systolic, mean and diastolic arterial pressures and heart rate were recorded every minute for the first 20 minutes. The nitrous oxide concentration was increased to 70% in oxygen for all patients after delivery of the infant and, in the thiopentone group, the enflurane concentration was reduced to 0.5%. An infusion of oxytocin (20 units in 500 ml 5% glucose) was started and morphine 0.2 mg/kg was given intravenously. The propofol infusion or the enflurane was discontinued at the start of skin suture and nitrous oxide stopped 2 minutes later. Residual neuromuscular block was antagonised with neostigmine 2.5 mg and atropine 1.2 mg.

Maternal recovery was timed from cessation of the propofol or enflurane and assessed by one investigator who was blind to the anaesthetic technique. The time that each patient took to open her eyes to command and the time to give her correct date of birth were recorded. Psychomotor recovery was assessed by the patient's ability to perform a modified 'postbox' test<sup>8</sup> in the left lateral position before operation and at 30 and 60 minutes after operation. The number of shapes that the patient could complete in one minute was recorded and the best of three attempts was accepted. All women were interviewed the next day about awareness.

Umbilical venous and arterial blood gas analysis were performed at delivery. All neonates were assessed by one paediatrician who was unaware of the anaesthetic technique. Apgar scores at one and five minutes, and Neurologic and Adaptive Capacity Scores (NACS)<sup>9</sup> at 15 minutes, 2 and 24 hours were recorded.

Student's *t*-test was used to compare demographic data and umbilical blood gas analysis while haemodynamic data were analysed by repeated measures analysis of variance. Maternal recovery, Apgar scores and NACS scores were compared using the Mann-Whitney test. Kendall Rank correlation was used to compare maternal recovery with anaesthetic duration and neonatal scores with induction to delivery times. Statistical significance was accepted when  $p \leq 0.05$ .

### Results

There were no demographic differences between the groups (Table 1). The quality of induction was difficult to assess because of the nature of rapid sequence induction. Lacrimation was noted in four patients in the propofol

Table 1. Demographic and anaesthetic data, mean (SD).

	Thiopentone	Propofol
Age; years	30.6 (5.1)	29.4 (4.3)
Height; cm	149 (12.1)	152 (6.9)
Weight; kg	65.4 (9.4)	65.8 (9.3)
Gestation; weeks	38.9 (1.3)	39.1 (1.5)
I-D time; minutes	12.2 (2.6)	10.8 (2.1)
Birth weight; kg	3.22 (0.62)	3.28 (0.43)
Anaesthetic time; minutes	48.2 (9.4)	42.0 (9.2)

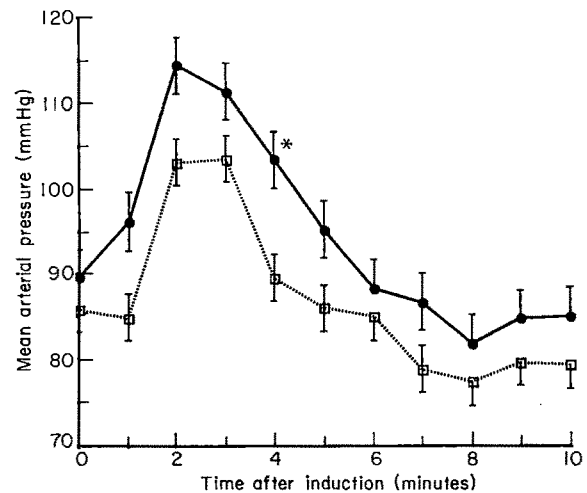


Fig. 1. Mean arterial pressure (SEM) after induction of anaesthesia in the thiopentone —●— and propofol ...□... groups. \*  $p < 0.05$  between groups.

group but not in the thiopentone group. No patients complained of pain on injection and intubation conditions were satisfactory in all cases.

The normal size adult blood pressure cuff was appropriate for all patients, and there were no significant changes in systolic and mean arterial pressures immediately after induction of anaesthesia compared with awake values. These increased after intubation in both groups ( $p < 0.01$ ) and returned to baseline levels by 4 minutes in the propofol group and 5 minutes in the thiopentone group (Fig. 1).

Maternal heart rate increased in both groups after induction of anaesthesia ( $p < 0.01$ ) and returned to baseline values by 7 minutes in the propofol group and 8 minutes in the thiopentone group (Fig. 2).

Total anaesthetic time was similar between groups, and ranged from 28 to 65 minutes. There were no differences between groups in the time to eye opening, time to correct date of birth or postbox scores. Recovery times were not correlated to total anaesthetic time (Table 2), and no patients complained of nausea or vomited.

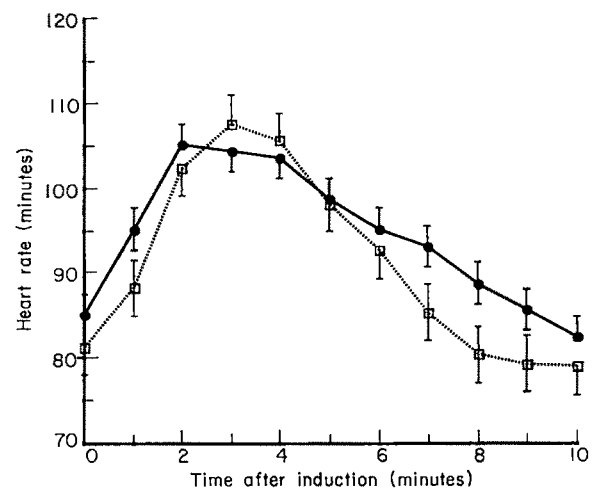


Fig. 2. Mean (SEM) maternal heart rate after induction of anaesthesia in the thiopentone —●— and propofol ...□... groups.

**Table 2.** Maternal recovery times and postbox scores, mean (SD).

	Thiopentone	Propofol
Eye opening; minutes	7.56 (3.0)	7.03 (2.5)
Date of birth; minutes	9.69 (3.2)	9.12 (2.5)
Postbox scores after 30 minutes compared with before operation	0.53 (0.27)	0.63 (0.15)
Postbox scores after 60 minutes compared with before operation	0.83 (0.24)	0.89 (0.19)

There were no differences in gestation or birth weight between the groups. Induction to delivery times were similar and uterine incision to delivery times were all less than 90 seconds.

Umbilical blood gas analysis, neonatal Apgar scores and NACS were satisfactory and similar between groups (Tables 3 and 4). Apgar scores and NACS were not related to induction-to-delivery times in the thiopentone group but NACS at 2 hours were slightly lower in the propofol group with increasing induction-to-delivery time ( $p < 0.05$ ) (Fig. 3).

Measured blood loss ranged from 200 to 1000 ml with no difference between groups. There were no anaesthetic or obstetric complications and no patients complained of awareness during anaesthesia.

### Discussion

The two anaesthetic techniques gave clinically satisfactory and similar results. There was no hypotension after induction of anaesthesia. The peak decrease in arterial blood pressure after propofol usually occurs at 2 to 3 minutes and patients in this study were subjected to the coincident stimuli of cricoid pressure and intubation. Arterial pressures, after tracheal intubation, returned to baseline levels more rapidly in the propofol group. Propofol attenuates the cardiovascular response to laryngoscopy and intubation more than thiopentone.<sup>1,10,11</sup>

Variations in arterial pressure and heart rate during Caesarean section are influenced more by the rapid circulatory volume changes during surgery and delivery than the anaesthetic technique. However, during other surgical procedures, the cardiovascular changes during maintenance of anaesthesia by propofol infusion have been comparable with the use of volatile agents.<sup>1,2,4</sup>

Maternal recovery was similar between groups although our previous study showed that a similar propofol group had slightly faster recovery times.<sup>6</sup> The anaesthetic technique differed slightly in this study since nitrous oxide was

**Table 4.** Apgar scores and neurologic and adaptive capacity scores (NACS), median (range).

	Thiopentone	Propofol
Apgar 1 minute	9 (5–10)	9 (5–10)
Apgar 5 minutes	10 (8–10)	10 (8–10)
NACS 15 minutes	32 (25–34)	32 (21–36)
NACS 2 hours	33 (23–35)	33.5 (25–36)
NACS 24 hours	33 (30–37)	34.5 (30–37)

discontinued 2 minutes after the propofol or enflurane, while the earlier study stopped all anaesthetic agents simultaneously. Propofol, after major operations, has not always shown faster recovery when compared with a volatile agent.<sup>1</sup>

No patients complained of awareness in this study, but with our sample size of 20 there is still a 5% probability that the incidence of awareness is as high as 14%. However, a previous study using the same propofol infusion rate measured propofol concentrations during anaesthesia and recovery, and in every patient propofol concentrations were higher during anaesthesia than at the time of eye opening.<sup>6</sup>

Inhalation anaesthetic requirements are decreased in pregnancy but this has not been investigated during intravenous anaesthesia. Minimum alveolar concentrations for inhalational agents are reduced in animal studies with pregnant ewes<sup>12</sup> and rats,<sup>13</sup> which may be related to increased levels of circulating endorphins.<sup>14</sup> There is, in pregnancy, decreased plasma binding of many drugs with increased free levels<sup>15</sup> but there are no data for propofol.

Comparative studies with thiopentone and propofol during induction of anaesthesia for Caesarean section have shown variable neonatal outcome. Apgar scores were similar<sup>11,16,17</sup> or lower after propofol.<sup>18,19</sup> Neurobehavioural assessment was satisfactory after propofol,<sup>5</sup> but other workers reported lower scores after propofol (2.8 mg/kg) compared with thiopentone (5 mg/kg).<sup>19</sup> In this study, NACS in the propofol group were slightly lower with longer I-D times. A longer infusion time before delivery is likely to cause higher neonatal levels at delivery, and lower NACS have been correlated with higher neonatal levels of propofol.<sup>20</sup> However, small differences in NACS have not been shown to have any clinical significance.

A propofol infusion coupled with nitrous oxide gave satisfactory induction and maintenance of anaesthesia. Haemodynamic changes at induction were less variable in the propofol group, compared with a thiopentone, nitrous oxide, enflurane technique, and neonatal effects were similar when infusion time before delivery ranged from 7 to

**Table 3.** Umbilical venous and arterial blood gas analysis, mean (SD).

	Thiopentone	Propofol
Umbilical venous pH	7.327 (0.028)	7.331 (0.056)
Umbilical venous $PCO_2$ (kPa)	5.63 (0.65)	5.89 (0.77)
Umbilical venous $PO_2$ (kPa)	4.35 (1.0)	3.80 (0.94)
Umbilical venous BE	-3.0 (1.9)	-2.8 (1.7)
Umbilical arterial pH	7.271 (0.038)	7.262 (0.034)
Umbilical arterial $PCO_2$ (kPa)	6.86 (1.0)	7.10 (0.91)
Umbilical arterial $PO_2$ (kPa)	2.44 (0.81)	2.19 (0.71)
Umbilical arterial BE	-3.0 (2.6)	-2.8 (2.2)

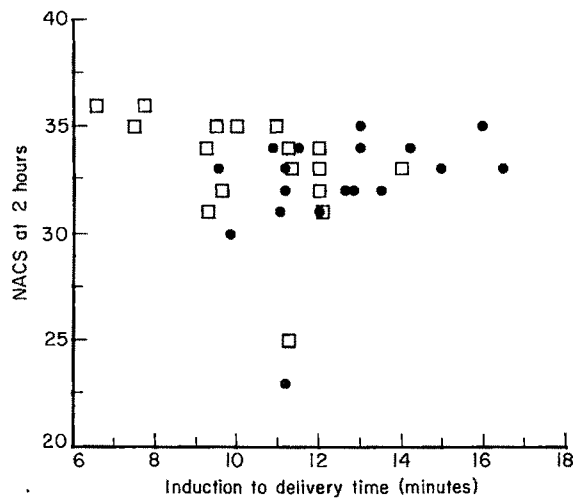


Fig. 3. Induction to delivery time (minutes) against neurologic and adaptive capacity scores 2 hours after delivery, ●, thiopentone group; □, propofol group.

14 minutes. However, a prolonged infusion time before delivery may cause slightly lower NACS scores. Maternal recovery was rapid and similar in both groups. We believe that maintenance of anaesthesia with inhalation agents remains the standard for obstetric general anaesthesia.

#### Acknowledgments

We thank Professor A. Chang, Chairman, Department of Obstetrics and Gynaecology for permitting this study on patients in his department, Mrs V. Kotur for her assistance and ICI Pharmaceuticals for their support.

#### References

- DOZE VA, SHAFFER A, WHITE PF. Propofol-nitrous oxide versus thiopental-iso-flurane-nitrous oxide for general anaesthesia. *Anesthesiology* 1988; **69**: 63-71.
- DE GROOT PMRM, HARBERS JBM, VAN EGMOND J, CRUL JF. Anaesthesia for laparoscopy. A comparison of five techniques including propofol, etomidate, thiopentone and iso-flurane. *Anaesthesia* 1987; **42**: 815-23.
- ZUURMOND WWA, VAN LEEUWEN L, HELMERS JHJH. Recovery from propofol infusion as the main agent for outpatient arthroscopy. A comparison with iso-flurane. *Anaesthesia* 1987; **42**: 356-9.

- SEAR JW, SHAW I, WOLF A, KAY NH. Infusions of propofol to supplement nitrous oxide-oxygen for the maintenance of anaesthesia. A comparison with halothane. *Anaesthesia* 1988; **43** (Suppl.): 18-22.
- DAILLAND P, COCKSHOTT ID, LIRZIN JD, JACQUINOT P, JORROT JC, DEVERY J, HARMEY J-L, CONSEILLER C. Intravenous propofol during caesarean section: placental transfer, concentrations in breast milk, and neonatal effects. A preliminary study. *Anesthesiology* 1989; **71**: 827-34.
- GREGORY MA, GIN T, YAU G, LEUNG R KW, CHAN K, OH TE. Propofol infusion anaesthesia for Caesarean section. *Canadian Journal of Anaesthesia* 1990; **37**: 514-20.
- GIN T, GREGORY MA, CHAN K, OH TE. Maternal and fetal levels of propofol at Caesarean section. *Anaesthesia and Intensive Care* 1990; **18**: 180-4.
- CRAIG J, COOPER GM, SEAR JW. Recovery from day-case anaesthesia. Comparison between methohexitone, Althesin and etomidate. *British Journal of Anaesthesia* 1982; **54**: 447-51.
- AMIEL-TISON C, BARRIER G, SHNIDER SM, LEVINSON G, HUGHES SC, STEFANI SJ. A new neurologic and adaptive capacity scoring system for evaluating obstetric medications in full-term newborns. *Anesthesiology* 1982; **56**: 340-50.
- HARRIS CE, MURRAY AM, ANDERSON JM, GROUNDS RM, MORGAN M. Effects of thiopentone, etomidate and propofol on the haemodynamic response to tracheal intubation. *Anaesthesia* 1988; **43** (Suppl.): 32-6.
- GIN T, GREGORY MA, OH TE. The haemodynamic effects of propofol and thiopentone for induction of Caesarean section. *Anaesthesia and Intensive Care* 1990; **18**: 175-9.
- PALAHNIUK RJ, SHNIDER SM, EGER, II, EI. Pregnancy decreases the requirement for inhaled anesthetic agents. *Anesthesiology* 1974; **41**: 82-3.
- STROUT CD, NAHRWOLD ML. Halothane requirement during pregnancy and lactation in rats. *Anesthesiology* 1981; **55**: 322-3.
- GINTZLER AR. Endorphin-mediated increases in pain threshold during pregnancy. *Science* 1980; **210**: 193-5.
- PERUCCA E, CREMA A. Plasma protein binding of drugs in pregnancy. *Clinical Pharmacokinetics* 1982; **7**: 336-52.
- VALTONEN M, KANTO J, ROSENBERG P. Comparison of propofol and thiopentone for induction of anaesthesia for elective Caesarean section. *Anaesthesia* 1989; **44**: 758-62.
- MOORE J, BILL KM, FLYNN RJ, MCKEATING KT, HOWARD PJ. A comparison between propofol and thiopentone as induction agents in obstetric anaesthesia. *Anaesthesia* 1989; **44**: 753-7.
- COUPER JL, LOMBARD TP. Comparison of propofol (Diprivan) with thiopentone as induction agent for elective Caesarean section. *Canadian Journal of Anaesthesia* 1988; **35**: S132.
- CELLENO D, CAPOGNA G, TOMASSETTI M, COSTANTINO P, DI FEO G, NISINI R. Neurobehavioural effects of propofol on the neonate following elective Caesarean section. *British Journal of Anaesthesia* 1989; **62**: 649-54.
- GIN T, YAU G, CHAN K, GREGORY MA, KOTUR CF. Recovery from propofol infusion anaesthesia for Caesarean section. *Neuroscience Letters* (Suppl.) 1990; **37**: S44.

## Ketamine as analgesic for total intravenous anaesthesia with propofol

J. B. M. GUIT, H. M. KONING, M. L. COSTER, R. P. E. NIEMEIJER AND D. P. MACKIE

### Summary

*A prospective study of 18 patients who underwent noncardiac surgery was performed to study the use of ketamine as an analgesic during total intravenous anaesthesia with propofol. A comparison was made with the combination propofol/fentanyl. The propofol/ketamine combination resulted in haemodynamically stable anaesthesia without the need for additional analgesics. Postoperative behaviour was normal in all patients and none of the patients reported dreaming during or after the operation. Propofol seems to be effective in eliminating side effects of a subanaesthetic dose of ketamine in humans. We recommend the propofol/ketamine combination for total intravenous anaesthesia for surgery when stable haemodynamics are required.*

### Key words

*Total intravenous anaesthesia.*

*Anaesthetics, intravenous; propofol, ketamine.*

Ketamine is a powerful analgesic, even in doses insufficient to induce anaesthesia.<sup>1</sup> It has many of the attributes of the 'ideal' analgesic agent for longer routine operations: a very high margin of safety, no irritation of the veins and no negative influence on ventilation or circulation. Its main disadvantages are that it produces hypertension and precipitates psychomimetic emergence phenomena. These effects can be mitigated by judicious medication, particularly by administering benzodiazepines. The combination of midazolam with ketamine has been recommended previously for total intravenous anaesthesia in military surgery, general civilian practice and cardiac surgery.<sup>2–4</sup>

In this study, the combination of propofol/ketamine was compared to the combination propofol/fentanyl in a double-blind, prospective trial in patients undergoing general anaesthesia for elective surgery. Haemodynamic variables, the time to recovery and patient acceptability were compared.

### Methods

A prospective study of 18 patients who underwent noncardiac surgery was performed. Patients gave informed consent to a protocol approved by the medical ethics committee of our hospital. All patients were ASA grade 1 or 2 and scheduled for operations longer than 15 minutes (Table 1). Patients received oral oxazepam (0.25–0.3

mg/kg) as premedication 2 hours before surgery and were allocated randomly to one of two groups to receive propofol with ketamine ( $n = 9$ ) or propofol with fentanyl ( $n = 9$ ) for total intravenous anaesthesia.

Standard lead II of the electrocardiogram was monitored and an intravenous cannula inserted on arrival of the patient in the operating theatre. Heart rate was detected by electrocardiogram and calculated electronically on a beat-to-beat basis. The pulse rate was timed for at least 30 seconds. One person recorded all arterial pressure measurements by auscultation (diastolic reading as Korotkoff phase V) using an anaeroid sphygmomanometer previously calibrated at zero and 150 mmHg against a mercury column. Anaesthesia was induced with propofol (2 mg/kg) and either fentanyl (3  $\mu$ g/kg) or ketamine (1 mg/kg). Vecuronium (0.15 mg/kg) was administered. Anaesthesia was maintained with propofol 12 mg/kg/hour during the first 30 minutes, followed by 9 mg/kg/hour for 30 minutes and then 6 mg/kg/hour combined with fentanyl 1.5  $\mu$ g/kg/hour or with ketamine 2 mg/kg/hour. The patient's lungs were ventilated with oxygen-enriched air with an  $F_{IO_2}$  of 0.35.

The postinduction arterial pressure and heart rate were recorded one minute after induction, and direct laryngoscopy with a curved blade was initiated 2 minutes after induction. None of the patients received topical or intravenous lignocaine before laryngoscopy, and tracheal intubation was always accomplished within 20 seconds.

J.B.M. Guit, MD, H.M. Koning, MD, M.L. Coster, MD, R.P.E. Niemeijer, MD, Department of Anaesthesia, Medical Centre Leeuwarden, Henri Dunantweg 2, 8934 AD Leeuwarden, The Netherlands, D.P. Mackie, FFARCS, Rode Kruis Ziekenhuis, Vondellaan 13, 1942 LE Beverwijk, The Netherlands.

Correspondence should be addressed to Dr H.M. Koning please.

Accepted 4 June 1990.

**Table 1.** The distribution of surgical procedures in the two groups.

	Propofol/fentanyl	Propofol/ketamine
Abdominal	1	2
Gynaecological	2	2
Orthopaedic	1	3
Plastic	3	1
Ear, nose and throat	2	1
Total	9	9

Arterial pressure and heart rate were recorded again one minute after tracheal intubation. Ventilation was set to achieve an end-tidal carbon dioxide concentration of 4% with a frequency of 12 breaths/minute. The tidal volume and the respiratory pressures were noted. The anaesthetist did not know whether fentanyl or ketamine had been used. Increments of analgesics were given during anaesthesia if analgesia was judged clinically to be adequate as assessed by: a sudden increase of systolic blood pressure of more than 20 mmHg; an increase in heart rate of more than 10 beats/minute, in the absence of hypovolaemia; and signs of sweating and lacrimation.

Additional vecuronium in a dose of 0.04 mg/kg was administered when muscle relaxation was judged to be inadequate. The continuous administration of drugs was stopped at the end of surgery and residual neuromuscular blockade was antagonised with intravenous atropine (0.01 mg/kg) and neostigmine (0.03 mg/kg). The time to awakening was noted. The trachea was extubated when the patient was able to maintain an adequate airway and good respiratory minute volume.

Arterial pressure, heart rate, the reaction of the patient to command and an assessment of whether the patient was awake or drowsy were noted in the recovery room by a nurse who was unaware of the type of anaesthesia. Five questions were asked 30 minutes after operation: the name of the patient, the date of birth, where the patient was, and the date and the time. Each patient was interviewed on the day after surgery by a doctor who was unaware whether fentanyl or ketamine was used. Patients were asked about side effects, awareness, and their opinion about the anaesthetic.

The Chi-square test was computed for dichotomous variables. The Wilcoxon rank sum test was used for continuous variables.<sup>5</sup> A p-value of less than 0.05 was considered significant.

### Results

The pre-operative variables in both groups were compared (Table 2). No statistically significant differences in gender,

age, height, weight, arterial pressure or heart rate were found.

The haemodynamic variables throughout the peri-operative period for both groups are shown in Figure 1. Stable arterial pressure and heart rate were seen in the patients who received propofol/ketamine, except for a temporary increase directly after tracheal intubation. Systolic pressure increased by 13%, diastolic pressure by 11% and heart rate by 14%.

Decreases in systolic and diastolic blood pressure were observed after induction in patients who received propofol/fentanyl. Arterial pressure returned to baseline values after intubation. The heart rate was stable, except for an increase in mean heart rate by 24% after intubation. Patients who received propofol/fentanyl tended to have a lower systolic pressure than patients who received propofol/ketamine.

The intra-operative variables for both groups are shown in Table 3. The lowest systolic pressure found in the patients with propofol/ketamine was statistically significantly higher than that observed in patients with propofol/fentanyl. No extra analgesics were required in the propofol/ketamine group, but patients who received propofol/fentanyl required a mean additional dose of fentanyl 0.72 µg/kg/hour. No statistically significant difference in the duration of surgery, respiratory variables, muscle relaxation or awakening after surgery were found.

The postoperative variables are shown in Table 4. Patients who received propofol/ketamine demonstrated a significantly longer recovery time than patients given propofol/fentanyl. The time to an adequate reaction and the duration of drowsiness were longer, and the responses to the five questions 30 minutes after surgery were significantly less adequate. There were increased incidences of dizziness and confusion in patients given propofol/ketamine, but in all cases the confusion was reported by the patient himself and judged to be minor. No confusion was observed by medical personnel. Postoperative behaviour was normal in all patients, and none reported dreaming during or after surgery. All patients judged the propofol/ketamine combination to be pleasant, compared to 89% of the patients with propofol/fentanyl.

**Table 2.** Pre-operative variables. Data are presented as mean (SD).

	Propofol-ketamine	Propofol-fentanyl
Females : males	7 : 2	6 : 3
Age; years	35 (12)	39 (12)
Height; cm	172 (10)	169 (6)
Weight; kg	70 (11)	72 (11)
Arterial pressure; mmHg		
Systolic	130 (11)	126 (11)
Diastolic	80 (10)	84 (9)
Heart rate; beats/minute	80 (16)	72 (9)

There were no significant differences.



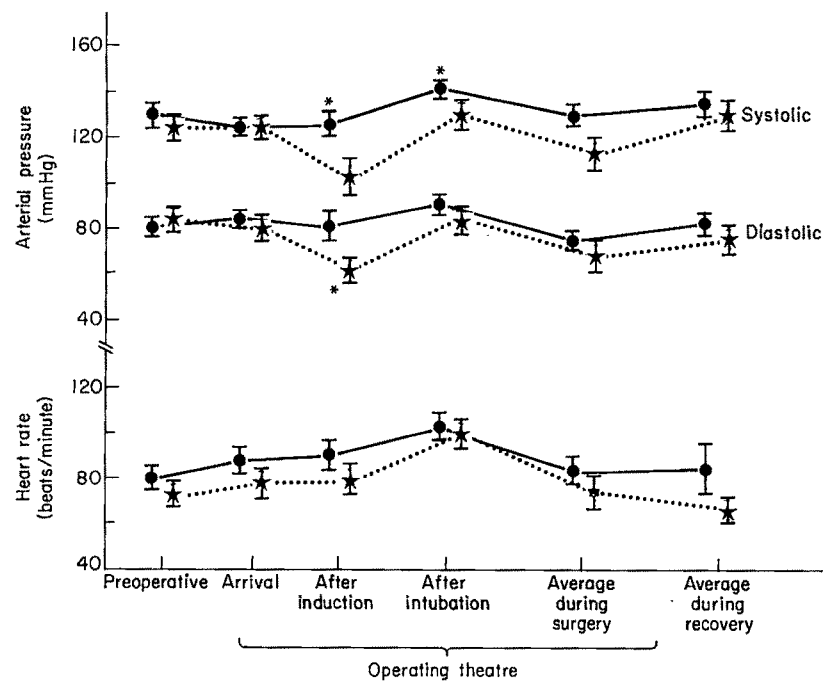


Fig. 1. Haemodynamic variables throughout the peri-operative period in patients anaesthetised with propofol/ketamine (●), or propofol/fentanyl (★). \*, statistical significance.

Table 3. Intra-operative variables. Data are presented as mean (SD).

	Propofol-ketamine	Propofol-fentanyl	p-value
Duration of surgery; minutes	60 (27)	78 (27)	ns
Respiratory minute volume; litres/minute	7.2 (0.5)	7.1 (1.1)	ns
Compliance; ml/kPa			
Static	6.2 (1.1)	6.0 (1.8)	ns
Dynamic	3.2 (0.4)	2.8 (0.7)	ns
Arterial pressure; mmHg			
Highest systolic	144 (12)	137 (19)	ns
Lowest systolic	118 (17)	99 (10)	0.030
Highest diastolic	85 (9)	81 (10)	ns
Lowest diastolic	64 (11)	59 (15)	ns
Heart rate; beats/minute			
Highest	92 (17)	81 (14)	ns
Lowest	73 (18)	66 (14)	ns
Muscle relaxation maintenance dose; mg/kg/hour	0.02 (0.02)	0.03 (0.03)	ns
Extra dose of fentanyl; µg/kg/hour	—	0.72 (0.72)	ns
Awakening after stopping TIVA; minutes	17 (5)	13 (18)	ns

TIVA, total intravenous anaesthesia.

ns, not significant.

Table 4. Postoperative variables. Data are presented as mean (SD).

	Propofol-ketamine	Propofol-fentanyl	p-value
Recovery-room			
Time until adequate reaction; minutes	19 (26)	1 (4)	0.027
Time to awakening; minutes	22 (23)	9 (10)	ns
Duration of drowsiness; minutes	36 (22)	11 (17)	0.036
Good response on five questions	3 (2)	5 (0)	0.024
Nausea; %	0	11	ns
Patients' interview			
Duration of awakening; hours	3.4 (2.7)	2.2 (3.0)	ns
Nausea; %	0	11	ns
Vomiting; %	0	11	ns
Dizzy; %	44	11	ns
Confused; %	33	11	ns
General judgement as good; %	100	89	ns

ns, not significant.

### Discussion

Ketamine in subanaesthetic doses has recently gained more attention as an analgesic for total intravenous anaesthesia. Ketamine is a powerful analgesic although the mechanism and site of action of its analgesic effect remain to be fully elucidated. It possesses local anaesthetic properties<sup>6</sup> and has a direct inhibitory action on the dorsal horn neurones of lamina I and V.<sup>7</sup> Antagonism of the analgesic effect of ketamine with naloxone in rats suggests that an endogenous opioid neuronal pathway in the central nervous system is involved.<sup>8</sup> However, an opioid component in the analgesic action of a subanaesthetic ketamine dose in humans has not been found, and blockade of the N-methyl-aspartate receptor has been suggested as an alternative mode of action.<sup>9</sup> N-methyl-aspartate receptors may represent a subgroup of the sigma opioid receptors which block spinal nociceptive reflexes. Several other neuronal systems may be involved in the antinociceptive action of ketamine, since blockade of the noradrenaline and serotonin receptor also attenuates the analgesic action of ketamine in animals.<sup>10</sup>

The advantages of the use of ketamine as an analgesic include a powerful action at small doses without myocardial or respiratory depression, a broad therapeutic range and no organotoxic effects. The cardiocirculatory stimulation includes increases in heart rate, cardiac index and arterial pressure. However, the high incidence of adverse reactions limits its use. The main adverse reactions are stimulation of the sympathetic nervous system resulting in hypertension and emergence reactions (delirium, nightmares, hallucinations), both of which cause problems in patient acceptance and in postoperative nursing care.

Various methods have been proposed to overcome the adverse reactions. The combination of ketamine with benzodiazepines reduces the incidence of side effects and a continuous infusion of midazolam with ketamine has been recommended for military surgery, general civilian practice and cardiac surgery.<sup>2-4</sup> Anaesthesia is characterised by stable haemodynamics, after a temporary initial increase in heart rate and systolic arterial pressure. Up to 15% of patients report dreaming.

The object of this study was to investigate whether propofol could eliminate the side effects of ketamine. The propofol/ketamine combination resulted in a stable arterial pressure and heart rate during surgery. Slow recovery was observed, but the postoperative behaviour was normal in all patients and no patient reported dreaming during or

after the operation. Propofol therefore seems to be effective in eliminating the side effects of a subanaesthetic dose of ketamine in humans.

The advantage of using ketamine as the analgesic in combination with propofol for total intravenous anaesthesia is the achievement of stable haemodynamics during surgery. A stable arterial pressure throughout the operative period was observed, compared with the lower blood pressure found in the patients given propofol/fentanyl. The maintenance dose of fentanyl was not sufficient for analgesia in all patients in the propofol/fentanyl group and a dose of 2.0 to 2.5 µg/kg/hour might have been more appropriate. However, a higher dose of fentanyl might have depressed the circulation even more. Side effects of the propofol/ketamine combination were the prolonged duration of the action of ketamine, resulting in a slower awakening and recovery, dizziness, and minor confusion directly after surgery. However, all patients judged anaesthesia to be good.

We recommend the propofol/ketamine combination for total intravenous anaesthesia when stable haemodynamics are warranted.

### References

1. REICH DL, SILVAY G. Ketamine. An update on the first twenty-five years of clinical experience. *Canadian Journal of Anaesthesia* 1989; **36**: 186-97.
2. RESTALL J, TULLY AM, WARD PJ, KIDD AG. Total intravenous anaesthesia for military surgery. A technique using ketamine, midazolam and vecuronium. *Anaesthesia* 1988; **43**: 46-9.
3. BAILIE R, CRAIG G, RESTALL J. Total intravenous anaesthesia for laparoscopy. *Anaesthesia* 1989; **44**: 60-3.
4. CHAI M, THANGA D, MORGAN E, VILJOEN J. Ketamine-midazolam continuous infusion in cardiac surgical patients. *Anaesthesia* 1989; **44**: 364.
5. NOETHER GE. *Introduction to statistics*. Boston: Houghton Mifflin, 1976.
6. AMIOT JF, BOUJOU PH, PALACCI JH, BALLINER E. Intravenous regional anaesthesia with ketamine. *Anaesthesia* 1985; **40**: 899-901.
7. KITAHATA LM, TAUB A, KOSAKA Y. Lamina-specific suppression of dorsal-horn unit activity by ketamine hydrochloride. *Anesthesiology* 1973; **38**: 4-11.
8. SMITH DJ, PEKOE GM, MARTIN LL, COALGATE B. The interaction of ketamine with the opiate receptor. *Life Sciences* 1980; **26**: 789-95.
9. MAURSET A, SKOGLUND LA, HUSTVEIT O, OYE I. Comparison of ketamine and pethidine in experimental and postoperative pain. *Pain* 1989; **38**: 37-41.
10. PEKOE GM, SMITH DJ. The involvement of opiate and monoaminergic neuronal systems in the analgesic effects of ketamine. *Pain* 1982; **12**: 57-73.

CASE REPORT

## Midazolam-induced benzodiazepine withdrawal syndrome

B. METS, A. HORSELL AND D. M. LINTON

### Summary

*A case history of a patient who developed severe anxiety and agitation on two occasions after discontinuation of a midazolam infusion is presented. The withdrawal symptoms interfered with effective mechanical ventilation and the patient required the reintroduction of a long-acting benzodiazepine to treat the withdrawal state and to facilitate weaning from mechanical ventilation.*

### Key words

*Hypnotics, benzodiazepines; midazolam.  
Complications; withdrawal syndrome.*

Midazolam, an ultrashort-acting water-soluble imidazo-benzodiazepine, is used as an intravenous sedative for patients whose lungs are ventilated in an ICU.<sup>1–3</sup> The rapid recovery from midazolam sedation was suggested as a likely cause of a benzodiazepine withdrawal state if a continuous infusion is discontinued abruptly.<sup>2</sup> We present a case history of a patient who we believe developed a clinical state of benzodiazepine withdrawal on two occasions during a complicated illness when a continuous midazolam infusion was abruptly discontinued.

### Case history

A 61-year-old man presented to the outpatients department of our hospital with a 4-day history of malaise, fever, nonproductive cough and increasing dyspnoea. He was found to be ill and toxic with a temperature of 40°C and had clinical, as well as radiological, evidence of right lower lobe pneumonia. He had a background history of severe uncontrolled hypertension and had suffered a cerebrovascular accident 8 years previously, leaving him with mild residual right-sided weakness. He neither smoked nor used alcohol or tranquillisers. The pneumonia was treated initially with intravenous penicillin and subsequently amikacin was added after 48 hours, when there was an obvious deterioration, with extension of the pneumonia and early acute renal tubular necrosis. The antibiotics were changed to erythromycin and ceftriaxone and he was transferred to the respiratory ICU. Initially he was managed by facemask with continuous positive airway pressure (CPAP), but

deteriorated further, so the trachea was intubated and he was started on IPPV.

All bacteriological cultures and serology studies were negative, despite an extending pneumonia and septicaemia. Management was further complicated by severe hypertension, fluid overload and secondary respiratory infection with *Klebsiella* Spp and *Pseudomonas* Spp. Furthermore, mechanical ventilation of the patient's lungs was difficult because of restlessness and tachypnoea, which caused marked arterial desaturation and exacerbation of hypertension. Sedation was required to improve ventilation. He was initially given two intravenous bolus doses of midazolam (15 mg) and subsequently a midazolam infusion was started at 3–5 mg/hour. In addition, he required occasional intravenous bolus doses of fentanyl (100 µg).

The midazolam infusion was stopped on day 6 (because it was expensive) and a morphine infusion started. Subsequently, the patient became extremely anxious, agitated and jittery. His agitated state persisted despite the addition of bolus doses of fentanyl, and resulted in ineffective mechanical ventilation; on day 11 a midazolam infusion was recommenced and was effective in improving ventilation. The infusion was stopped once again on day 16 and the same clinical picture developed. There were no signs of opioid withdrawal on either occasion. It was decided not to re-introduce midazolam, and a variety of agents were tried over the next 4 days, including a fentanyl infusion (0.3–0.5 mg/hour), intravenous clothiapine (40–80 mg, 8 hourly) and haloperidol (10 mg, 12 hourly).

The patient's agitated and restless state by day 21 was a

B. Mets, MB, ChB, FFARCS(Eng), FFA(SA), Lecturer in Clinical Pharmacology, Specialist Anaesthetist, A. Horsell, MB, ChB, Senior Medical House Officer, D.M. Linton, MB, ChB, FFA(SA), Senior Specialist Anaesthetist and Senior Lecturer, Respiratory Intensive Care Unit. University of Cape Town and Groote Schuur Hospital. Observatory 7925, South Africa.  
Accepted 21 December 1989.

major problem which prevented weaning from artificial ventilation.

A diagnosis was made of benzodiazepine withdrawal. All other sedatives and narcotics were withdrawn and he was managed with diazepam, initially with an intravenous bolus dose of 10 mg, and then 10 mg 8 hourly by nasogastric tube. The doses were gradually decreased over the next 2–3 weeks. This strategy proved entirely successful. The patient was weaned without any further major problems and the diazepam was stopped after 3 weeks. The patient made a full recovery and was discharged home.

### Discussion

A withdrawal syndrome after the use of benzodiazepines was first described by Hollister *et al.*<sup>4</sup> Its manifestations vary from the subtle (i.e. increased levels of anxiety and insomnia) to the more obvious signs of psychomotor agitation, with muscle twitching, sweating and the possibility of convulsions.<sup>5</sup>

In the complex clinical setting of an ICU it is difficult to establish the aetiology of severe agitation and in this patient an opioid withdrawal state, an organic confusional state and the possibility of an 'ICU psychosis' were considered. There were no specific features of an opioid withdrawal state, such as lacrimation, dilated pupils, gastrointestinal disturbances, muscle twitching or goose flesh. Specific organic causes of confusion such as hypoxaemia, metabolic derangement and electrolyte abnormalities were corrected or excluded. The temporal relationship of the onset of agitation after midazolam withdrawal, as well as the dramatic response to its restoration, and the subsequent change to diazepam, point strongly to benzodiazepine withdrawal as the main aetiological factor in the patient.

There is a paucity of literature about this syndrome in association with the intravenous administration of benzodiazepines, despite their extensive use as sedatives in intensive care. In this context, diazepam and lorazepam are the most commonly used.<sup>6,7</sup> The lack of reports may be as a result of the failure to recognise this syndrome in the ICU. It is more likely, in the case of diazepam, that after prolonged administration this agent has an extended time course in the body because of the production of long acting metabolites<sup>6,8</sup> and therefore avoids precipitating its own withdrawal symptoms.

Conversely, it appears that lorazepam which is not metabolised to active metabolites and has an intermediate duration of action (half-life: 7–11 hours) may occasionally produce a withdrawal syndrome. Simpson and Eltringham<sup>8</sup> in a study of 36 ICU patients, noted postsedation confusion associated with the use of lorazepam in two patients. It is possible that these patients suffered from benzodiazepine withdrawal.

Midazolam would appear to be an ideal agent for use in the ICU since its rapid elimination half-life of approximately 2 hours<sup>9</sup> allows titration to a suitable level of sedation, whilst the venous sequelae associated with diazepam do not occur.<sup>8</sup>

However, in the development of benzodiazepines such as midazolam with shorter elimination half-lives and therefore fewer hangover effects, there is an increasing awareness of the possibility of acute withdrawal from these agents after

short-term use.<sup>9</sup> The shorter acting the agent, the more rapid the onset and the more severe the withdrawal syndrome.<sup>10</sup> Triazolam serves as a good illustration since there is well documented evidence that acute withdrawal states occur after night-time use of this agent.<sup>9</sup>

Similarly it can be anticipated that midazolam, which is a potent agent with an even shorter elimination profile than triazolam (half-life, 4 hours)<sup>11</sup> and is used in the far higher dose range required in intensive care, may precipitate severe withdrawal reactions. The 'triazolam model' may also help to explain the exceedingly high doses of midazolam, described as 'midazolam resistance',<sup>3</sup> occasionally required in ICU.

This phenomenon may not necessarily be the result of acute tolerance. An alternative hypothesis may be that with intermittent administration and also during the course of continuous infusion (where fluctuations in plasma concentration are observed)<sup>12</sup> acute withdrawal reactions may occur. Such reactions may be misinterpreted as the result of inadequate sedation and higher dosages of midazolam are then prescribed. In this way a vicious cycle is engendered.

Midazolam is theoretically a good agent for use in the ICU because of its potency and ultrashort half-life. However, the experience of our patient indicates that this drug may lead to severe benzodiazepine withdrawal reactions if it is discontinued abruptly; this could limit its use as a long-term sedative in intensive care.

### Acknowledgments

The authors thank Dr A. H. Robins and Professor P. D. Potgieter for reviewing the manuscript.

### References

1. REVES JG, FRAGEN RJ, VINIK RH, GREENBLATT DJ. Midazolam: Pharmacology and uses. *Anesthesiology* 1985; **62**: 310–24.
2. FINLEY PR, NOLAN PE. Precipitation of benzodiazepine withdrawal following sudden discontinuation of midazolam. *Drug Intelligence and Clinical Pharmacy* 1989; **23**: 151–2.
3. SURY MRJ, BILLINGHAM I, RUSSELL GN, HOPKINS CS, THORNINGTON R, VIVORI E. Acute Benzodiazepine withdrawal syndrome after midazolam infusions in children. *Critical Care Medicine* 1989; **17**: 301–2.
4. HOLLISTER LE, MOTZENBECKER FP, DEGAN RO. Withdrawal reactions from chlorthalidopoxide. *Psychopharmacologia* 1963; **2**: 63–8.
5. OWEN RT, TYRER P. Benzodiazepine dependence. A review of the evidence. *Drugs* 1983; **25**: 385–98.
6. DOBB GJ, MURPHY DF. Sedation and analgesia during intensive care. *Clinics in Anaesthesiology* 1985; **3**: 1055–85.
7. MERRIMAN HM. The techniques used to sedate ventilated patients. *Intensive Care Medicine* 1981; **7**: 217–24.
8. SIMPSON PJ, ELTRINGHAM R. Lorazepam in intensive care. *Clinical Therapeutics* 1981; **4**: 150–63.
9. MORGAN K, OSWALD I. Anxiety caused by a short-life hypnotic. *British Medical Journal* 1982; **284**: 942.
10. BUSTO U, SELLERS EM, NARANJO CA, CAPPEL H, SANCHEZ-CRAIG M, SYKORA C. Withdrawal reaction after long-term therapeutic use of benzodiazepines. *New England Journal of Medicine* 1986; **315**: 854–9.
11. TYRER P. Dependence as a limiting factor in the clinical use of minor tranquilisers. *Pharmacology and Therapeutics*. 1988; **36**: 173–88.
12. KLOTZ U, REIMANN IW. Chronopharmacokinetic study with prolonged infusion of midazolam. *Clinical Pharmacokinetics* 1984; **9**: 469–74.

CASE REPORT

## Response to suxamethonium in a myasthenic patient during remission

M. ABEL, J. B. EISENKRAFT AND N. PATEL

### Summary

*A cumulative dose followed by an infusion was used to determine the dose response to suxamethonium in a patient with diagnosed myasthenia gravis who was in true remission (asymptomatic while receiving no therapy). The  $ED_{50}$  and  $ED_{90}$  values for suxamethonium were 0.08 mg/kg and 0.20 mg/kg, and an infusion rate of 3.2 mg/kg/hour was required to maintain a 90–95% depression of the single twitch response as monitored by integrated electromyography. These values are within the range for normal patients, and we conclude that myasthenic patients during a true remission may not demonstrate resistance to suxamethonium.*

### Key words

*Complications; myasthenia gravis.*

*Measurement techniques; electromyography, neuromuscular blockade.*

*Neuromuscular relaxants; suxamethonium.*

Myasthenia gravis is a chronic neuromuscular disorder characterised by weakness and fatigue of voluntary muscles with improvement after rest. It is also a disease of relapses and remissions.<sup>1</sup> Patients with active disease show increased sensitivity to the effects of nondepolarising neuromuscular blocking agents<sup>2,3</sup> and resistance to suxamethonium.<sup>4,5</sup> The responses of those patients to nondepolarising agents during a remission are controversial and poorly documented.<sup>6–9</sup> We describe a patient with myasthenia gravis who, during remission from his disease, demonstrated a normal response to the neuromuscular blocking effects of suxamethonium. There is no report in the literature to our knowledge about the responses to suxamethonium during a true remission.

### Case history

A 46-year-old, 100-kg male was admitted for laryngoscopy and vocal cord biopsy. He had been well until 9 years before admission when a mass had developed in his throat; this was treated with prednisone. The prednisone was discontinued 2 years later and the patient developed generalised weakness of his arms and legs, diplopia, but no bulbar symptoms. Myasthenia gravis was diagnosed by electromyographic (EMG) testing and was treated with oral pyridostigmine 90 mg five times daily. He underwent transcervical thymectomy 2 months later, after which he

required a brief period of postoperative mechanical ventilation of the lungs. Treatment with pyridostigmine and plasmapheresis was continued for a further 2 years until the patient went into remission; he became asymptomatic without any therapy for myasthenia gravis.

The neck mass was diagnosed as a lymphoblastic lymphoma 6 years before the present admission, and the patient received courses of chemotherapy consisting of cyclophosphamide, bleomycin, adriamycin and prednisone in the 2 years before the present admission. His last course was completed 4 months before admission. The patient had a 6-month relapse of myasthenia 16 months before admission which was treated by pyridostigmine 60 mg five times daily. For the 10 months immediately before the present admission he was asymptomatic and not taking any pyridostigmine, that is, he was in true remission.

All of the patient's routine laboratory test results, except CPK, were within normal limits on this admission (Hb 13.3 g/dlitre; Hct 41%;  $Na^+$  141 mEq/litre;  $K^+$  4.0 mEq/litre;  $Cl^-$  105 mEq/litre;  $HCO_3^-$  27 mEq/litre; glucose 93 mg/dlitre;  $Ca^{2+}$  8.8 mmol/litre; SGPT 41 u/litre (normal = 1–53); CPK 244 u/litre (normal = 25–145); alkaline phosphatase 48 u/litre (normal = 30–110); PT 12.3/12.2 seconds; PTT 27.8/31.3 seconds; bilirubin 0.4 mg/dlitre; albumin 4.0 g/dlitre; total protein 7.8 g/dlitre.

The patient received oral diazepam 10 mg one hour before the scheduled laryngoscopy and biopsy. Surface

M. Abel, MD, Resident, J.B. Eisenkraft, BSc, MD, MRCP, FFARCS, Professor, N. Patel, MB, BS, FFARCS, Assistant Professor, Department of Anesthesiology, Mount Sinai Medical Center, Box 1010, One Gustave L. Levy Place, New York, New York, NY 10029–6574, USA.

Reprint requests should be addressed to Professor Eisenkraft please.

Accepted 22 February 1990.



electrodes for neuromuscular transmission monitoring using a Datex 221 integrated EMG monitor (Datex/Puritan Bennett Co., Wilmington, Massachusetts) were applied over the ulnar nerve at the wrist and over the first dorsal interosseous muscle. General anaesthesia was induced with thiopentone,  $N_2O$ , and  $O_2$ , and maintained with supplemental doses of fentanyl and thiopentone, after a tracheostomy had been performed under local anaesthesia. No inhalation anaesthetic was administered. Ventilation was assisted to maintain the end-tidal  $CO_2$  between 35 and 40 mmHg.

The EMG monitor was calibrated, and trains of four (ToF) supramaximal stimuli (2 Hz) were applied to the ulnar nerve every 10 seconds while the integrated EMG response of the first dorsal interosseous muscle was recorded continuously. The patient was given an intravenous bolus of suxamethonium 0.10 mg/kg when the EMG response was stable, followed by intermittent boluses of 0.05 mg/kg, together with an infusion of suxamethonium, the rate of which was adjusted to replace drug which was calculated to have been eliminated.<sup>10</sup> Relaxation for the rest of the 15-minute surgical procedure was maintained using the suxamethonium infusion once 90–95% depression of the first response of the ToF compared with control (single twitch) had been achieved. Recovery from neuromuscular block was monitored after stopping the infusion. A blood sample was drawn for estimation of plasma cholinesterase activity and dibucaine number after the procedure.

A linear regression was obtained between the logarithm of the cumulative dose of suxamethonium and the logit transformation of neuromuscular block as measured by a percentage decrease in the single twitch height. The  $ED_{50}$ ,  $ED_{95}$ , and slope were calculated from this individual dose-response curve.

### Results

There were no complications of anaesthesia or surgery. The infusion of suxamethonium provided adequate relaxation for surgery once a single twitch value between 5 and 10% of control had been attained. There were no signs of phase II block (defined as train-of-four ratio < 0.5) at any time during the suxamethonium infusion or during recovery. The duration of the suxamethonium infusion was 19 minutes and the total dose administered was 85 mg. The calculated  $ED_{50}$  was 0.08 mg/kg and the  $ED_{95}$  was 0.20 mg/kg. A mean suxamethonium infusion rate of 3.2 mg/kg/hour was required to maintain 90–95% single twitch depression. The 25–75% recovery index was 10.3 minutes and the 10–90% recovery index was 24 minutes. The dibucaine number was 89 and the plasma cholinesterase level was 1740 milli units/ml (normal range 1900–3800 milli units/ml).

### Discussion

The responses to both depolarising and nondepolarising muscle relaxants of myasthenia gravis patients with active disease are well documented,<sup>3,4</sup> but sparse and conflicting data are available during states of remission. Both sensitivity,<sup>7,9</sup> and normal responses,<sup>8,18</sup> to nondepolarising relaxants have been described in myasthenic patients during remission. No such data are, however, available for suxamethonium.

We used the cumulative dose plus infusion technique

described by Smith *et al.*<sup>10</sup> in order to derive potency estimates for suxamethonium. This technique has the advantage that a complete dose-response relationship can be obtained in an individual patient, and the ED values derived may be compared with other published data.<sup>11</sup> The technique originally described used mechanomyography (MMG),<sup>10</sup> but we used an integrated EMG monitor, since it was found that there is a linear correlation between the two types of responses (EMG and MMG) during onset of and recovery from suxamethonium neuromuscular block.<sup>12</sup> Smith *et al.*<sup>13</sup> compared MMG and EMG responses of the *adductor pollicis* to suxamethonium in five normal patients and found an excellent correlation between the two methods ( $r = 0.97$ ). Potency data (ED values) derived for suxamethonium using the two methods were essentially identical.<sup>13</sup>

Myasthenics in relapse, are reported to be resistant to suxamethonium.<sup>4,5</sup> The  $ED_{50}$  and  $ED_{95}$  values of 0.08 mg/kg and 0.20 mg/kg in our patient are lower than the mean values (95% confidence limits in parentheses) of 0.33 (0.22–0.54) mg/kg and 0.82 (0.45–1.48) mg/kg, respectively, reported for myasthenics with active disease,<sup>4</sup> which indicates that our patient did not demonstrate resistance to suxamethonium. The mean (95% confidence limits in parentheses)  $ED_{50}$  and  $ED_{95}$  values obtained in 10 normal patients using an identical methodology were 0.17 (0.15–0.20) and 0.31 (0.27–0.37) mg/kg, respectively.<sup>4</sup> Others<sup>13</sup> have reported mean (SEM)  $ED_{50}$  and  $ED_{95}$  values of 0.12 (0.01) and 0.18 (0.01) mg/kg, respectively, in five normal patients when using a similar technique (integrated EMG but not the Datex monitor). It is likely that the differences in mean values between these two studies in normal individuals<sup>4,13</sup> merely represent variability in small samples. We therefore consider that the  $ED_{50}$  and  $ED_{95}$  values obtained in our patient fall within the range for normal patients.

The suxamethonium infusion rate required to maintain 90–95% single twitch EMG depression in our patient was 3.2 mg/kg/hour, which is within the normal range of 1.7–15.2 mg/kg/hour.<sup>14</sup> The 10–90% recovery index of 24 minutes represented a prolongation of recovery. Walts and Dillon<sup>15</sup> reported mean 10–90% recovery indices of 2.7–4.7 minutes following single intravenous suxamethonium doses of 0.5–4.0 mg/kg in patients without phase II block. Katz and Ryan<sup>14</sup> described large patient variability in recovery index, recovery of mechanomyographic single twitch from 10 to 90% of control ranging from 3–21 minutes following continuous suxamethonium infusion. These authors<sup>14</sup> did not, however, distinguish between patients with and without phase II block. Our patient did not demonstrate signs of phase II block.

Postoperative blood analysis revealed a normal dibucaine number but a slightly decreased plasma cholinesterase activity; the latter may have contributed to the slightly prolonged recovery. Howland and Smith<sup>16</sup> found that cyclophosphamide-treated patients had lower plasma cholinesterase levels than patients treated with other chemotherapy, but they did not report either the total dose of cyclophosphamide or the time between termination of therapy and blood sampling. Our patient had received several courses of chemotherapy, including cyclophosphamide; the most recent course was completed 4 months before the present surgical procedure.

There is currently little information on the morphology of the neuromuscular junction during remission from

myasthenia gravis, but it is known that the number of acetylcholine receptors is decreased in patients with active disease.<sup>17</sup> An increase in the receptors may account for our patient's apparently normal response to suxamethonium if the number of receptors changes with changing status of the myasthenia gravis.

In conclusion, we report one patient with myasthenia gravis in true remission (asymptomatic while receiving no therapy) who demonstrated a normal response to suxamethonium. Some have suggested that myasthenic patients in remission respond normally to nondepolarising relaxants,<sup>8,18</sup> while others<sup>7,9</sup> have suggested that symptomless myasthenic patients should not be regarded as 'cured'. Clearly, further studies of such patients would be needed to make any statement on their responses during a remission. Such an investigation would be difficult because of the small number of patients available for study.

### References

1. DRACHMAN DB. Myasthenia gravis-Part II. *New England Journal of Medicine* 1978; **298**: 186-93.
2. FOLDES FF, GLASER GH. Diagnostic tests in myasthenia gravis: an overview. *Annals of the New York Academy of Sciences* 1971; **183**: 275-86.
3. FOLDES FF, McNALL PG. Myasthenia gravis: a guide for anesthesiologists. *Anesthesiology* 1962; **23**: 837-72.
4. EISENKRAFT JB, BOOK WJ, MANN SM, PAPATESTAS AE, HUBBARD M. Resistance to succinylcholine in myasthenia gravis: a dose-response study. *Anesthesiology* 1988; **69**: 760-3.
5. WAINWRIGHT AP, BRODERICK PM. Suxamethonium in myasthenia gravis. *Anaesthesia* 1987; **42**: 950-7.
6. GRIGGS RC, McFARLIN DE, ENGEL WI. Severe occult juvenile myasthenia gravis responsive to long-term corticosteroid therapy. *Transactions of the American Neurological Association* 1968; **93**: 216-8.
7. LAKE CL. Curare sensitivity in steroid treated myasthenia gravis: a case report. *Anesthesia and Analgesia... Current Researches* 1978; **57**: 132-4.
8. FILLMORE KB, HERREN AL, PIRLO AF. Curare sensitivity in myasthenia gravis. *Anesthesia and Analgesia... Current Researches* 1978; **57**: 515-6.
9. LUMB AB, CALDER I. 'Cured' myasthenia gravis and neuromuscular blockade. *Anaesthesia* 1989; **44**: 828-30.
10. SMITH CE, DONATI F, BEVAN DR. Dose-response curves for succinylcholine: single versus cumulative techniques. *Anesthesiology* 1988; **69**: 338-42.
11. SMITH CE, LEWIS G, DONATI F, BEVAN DR. Dose-response relationship for succinylcholine in a patient with genetically-determined low plasma cholinesterase activity. *Anesthesiology* 1989; **70**: 156-8.
12. WEBER S, MURAVCHIK S. Electrical and mechanical train-of-four responses during depolarising and non-depolarising neuromuscular blockade. *Anesthesia and Analgesia* 1986; **65**: 771-6.
13. SMITH CE, DONATI F, BEVAN DR. Potency of succinylcholine at the diaphragm and at the *adductor pollicis* muscle. *Anesthesia and Analgesia* 1988; **67**: 625-30.
14. KATZ RL, RYAN JF. The neuromuscular effects of suxamethonium in man. *British Journal of Anaesthesia* 1969; **41**: 381-90.
15. WALTZ LF, DILLON JB. Clinical studies on succinylcholine chloride. *Anesthesiology* 1967; **28**: 372-6.
16. HOWLAND WS, GOLDINER PL. Physiologic management of cancer patients during surgery. In: HICKEY RC, ed. *Current problems in cancer*, Vol. III, no. 3. Chicago: Year Book Medical Publishers 1978; 12-13.
17. FAMBROUGH DM, DRACHMAN DB, SATYMURTI S. Neuromuscular junction in myasthenia gravis: decreased acetylcholine receptors. *Science* 1973; **182**: 293-5.
18. AZAR I. The response of patients with neuromuscular disorders to muscle relaxants: a review. *Anesthesiology* 1984; **61**: 173-87.

CASE REPORT

## Emery–Dreifuss muscular dystrophy

P. MORRISON AND R. H. JAGO

### Summary

*Emery–Dreifuss syndrome is a rare form of muscular dystrophy associated with cardiac complications that lead to sudden death. The disorder and its potential anaesthetic implications in the management of a patient who presented for orthopaedic surgery is described.*

### Key words

*Anaesthesia; techniques.*

*Complications; Emery–Dreifuss muscular dystrophy.*

Dreifuss and Hogan<sup>1</sup> in 1961 reported a rare, benign variant of Duchenne muscular dystrophy. In 1966 Emery and Dreifuss<sup>2</sup> defined the unique features that made it distinct from the Duchenne and Becker forms of the disorder (Table 1). This variant, in retrospect, may well have been described in two affected brothers as long ago as 1902.<sup>3</sup> The term Emery–Dreifuss muscular dystrophy (EDMD) was applied to the syndrome by Rowland *et al.*<sup>4</sup> in 1979, and is characterised by the triad of: early contractures of the elbows, Achilles tendons and posterior cervical muscles; slowly progressive muscle wasting and weakness with a humero-peroneal distribution in the early stages; a cardiomyopathy usually presenting as heart block.

It has since become clear that the clinical manifestations of both the neuromuscular and cardiac disorders encompass a somewhat broader spectrum<sup>5</sup> and that although the former runs a relatively benign course the latter may result in early sudden death<sup>6–8</sup> unless a cardiac pacemaker is inserted.<sup>9</sup>

X-linked inheritance is the typical pattern, with the responsible gene linked to colour blindness<sup>10</sup> and to DNA markers located around Xq27/28 at the distal end of the long arm of the x chromosome.<sup>11–13</sup> Sporadic cases have also been identified,<sup>7</sup> as has autosomal dominant transmission with occurrences in females.<sup>14,15</sup> The latter was labelled Emery–Dreifuss muscular dystrophy phenotype (EDMDP).<sup>5</sup>

EDMD usually presents at the age of 4 to 5 years with flexion contractures. Usually no significant muscle weak-

ness can be elicited at this time. Elbow involvement results in semi-flexed arms, and tightening of the Achilles tendons causes patients to walk on their toes. Limitation of neck flexion may go unnoticed at this stage. Muscle weakness and wasting gradually develops over the next decade. Initially the biceps, triceps (humero-) and anterior tibial and peroneal muscles (-peroneal) are affected, followed by hip and knee extensors and the proximal upper limb musculature (scapulo-humero-pelvo-peroneal). Progression of the disease is very slow and patients rarely lose the ability to walk. The early stages of muscle wasting are associated with a moderate increase in serum creatine phosphokinase (3–10 times normal), but this never approaches the levels found in Duchenne muscular dystrophy. Cardiac symptoms usually develop during the third decade of life. Syncopal attacks occur and frank heart block may result in sudden death. Electrocardiographic evidence of sinus bradycardia and prolongation of the P-R interval may be demonstrated before onset of symptoms.

### Case history

A 22-year-old hod-carrier presented for lengthening of both Achilles tendons. He had been referred to a neurologist, 18 months previously, with a 2-year history of increasing symmetrical proximal weakness of his legs and a vague, ill-defined pain in both thighs, hamstrings and left ankle. He also described occasional, brief episodes of paroxysmal tachycardia. He stated that his mother had

P. Morrison, MB, BS, DA, Registrar, R.H. Jago, MA, MB, BChir, FFARCS, Consultant, Department of Anaesthesia, Royal Berkshire Hospital, London Road, Reading, Berkshire RG1 5AN.

Accepted 22 February 1990.

Table 1. Distinguishing features of the x-linked muscular dystrophies.

Type	Age of onset	Course	Calf pseudo hypertrophy	Cardiomyopathy	Contractures
Duchenne	5	Severe	+	+	+
Becker	5-25	Benign	+	?	-
Emery-Dreifuss	4-5	Benign	-	+	+

died suddenly in her sleep in her early 30s and that she was wheelchair bound for the last years of her life. He gave no other positive family history.

General medical examination was unremarkable. He was a fit young man of normal mental and physical state. Blood pressure was 130/60 mmHg and he was in sinus rhythm. Bilateral ankle contractures were present, as were appearances suggestive of pes cavus. There was no facial myopathy, but his shoulder girdles (particularly the spinati), deltoid and pectoral muscles were wasted, and there was partial winging of the scapulae. His quadriceps were also wasted, but no peroneal wasting or calf hypertrophy was demonstrated.

Investigation revealed significant elevation of the plasma CPK at 2600 IU (normal 50-200 IU) and a slightly raised ALT of 56 IU (normal < 50 IU) and uric acid at 0.52 mmol/litre (normal 0.2-0.45 mmol/litre). All other blood tests were within normal limits. Electromyography confirmed primary muscle disease with normal motor and sensory nerve conduction. Muscle biopsy revealed moderately severe changes suggestive of chronic myopathy with type 1 fibres predominating. A resting ECG showed an unusual degree of right axis deviation with a P-R interval at the upper limit of normal. A 24-hour ambulatory ECG showed no arrhythmias, and lung function tests were within normal limits. A diagnosis of EDMD was made on the basis of this history, clinical examination and special investigation results, and the patient was referred to an orthopaedic surgeon for elongation of his Achilles tendons in order to facilitate walking.

At the pre-operative visit the history and findings were confirmed and the absence of previous exposure to anaesthesia established. After consideration of the risks associated with general anaesthesia in the presence of muscular dystrophy and detailed discussion with the patient and surgeon it was decided to proceed under subarachnoid block.

The patient was not premedicated. The ECG and temperature were monitored in the anaesthetic room and a preload of haemaccel solution 500 ml was given by intravenous line. Lumbar puncture was performed between the third and fourth lumbar vertebrae with a 25-gauge Becton-Dickinson spinal needle and 3 ml 0.5% bupivacaine was injected without barbotage. Loss of all sensation to the level of the 10th thoracic vertebra occurred within 4 minutes and bilateral arterial tourniquets were applied at mid-thigh level after exsanguination of the lower limbs. The patient was moved into the operating theatre, placed in the prone position and surgery completed in 35 minutes.

Pulse, blood pressure and temperature remained constant throughout the operation, after the initial decrease in systolic blood pressure associated with the onset of spinal block. The patient was transferred to the recovery room at the end of the procedure where monitoring was continued until the block began to regress. He

was returned to the ward at this stage. A halothane-free anaesthetic machine, dantrolene sodium and the facility for inserting an intracardiac pacing wire were held in constant readiness throughout this whole period. Subsequent recovery and mobilisation were uneventful.

### Discussion

The authors believe that this case represents another example of the EDMD phenotype despite the neurological diagnosis. His mother clearly suffered from muscle weakness and her sudden, unexplained death is likely to have been a consequence of her cardiomyopathy. Obligate female carriers of the x-linked form were reported to suffer from nonspecific cardiac symptoms with a substantial risk of serious sequelae,<sup>7,16</sup> but were not noted to express the myopathic symptoms of the syndrome. Unfortunately our patient was unable to give a more complete family history and we were unable to trace any further details of his mother's death.

EDMD is a rare disorder; only 177 cases have so far been reported,<sup>17</sup> and the autosomal dominant phenotype is even less common.<sup>15</sup> We immediately reviewed the standard anaesthetic reference texts when this patient presented for anaesthesia, including Katz, Benumof and Kadis,<sup>18</sup> and were unable to find any mention of the syndrome. A search of the literature failed to reveal any reference to interactions between the disease and anaesthesia, despite the fact that many cases had undergone surgery and presumably received some form of anaesthetic.<sup>2,7-9</sup>

This could mean that no complications were encountered. However, Smith and Bush's detailed review of the problems associated with anaesthesia and the other progressive muscular dystrophies (Duchenne and Becker) suggest that this is unlikely to be the case;<sup>19</sup> even allowing for the more benign progress of the skeletal myopathy, the potential for similar problems must exist in the Emery-Dreifuss form. They summarised the potential difficulties as follows: respiratory muscle weakness with inability to cough causing accumulation of secretions; hypomotility of the stomach with delayed gastric emptying associated with depressed laryngeal reflexes that contribute to an increased risk of aspiration; postoperative acute gastric dilatation; myocardial involvement resulting in tachyarrhythmias, ventricular fibrillation and cardiac arrest; malignant hyperpyrexia symptoms after suxamethonium and halothane; and delayed muscle weakness leading to postoperative respiratory failure after the reversal of nondepolarising muscle relaxants. Perioperative cardiac complications in Duchenne patients were also attributed to hyperkalaemia secondary to rhabdomyolysis.<sup>20,21</sup> Buzello and Huttarsch<sup>22</sup> reported up to six-fold delays in the recovery times of twitch tension after the use of vecuronium in Duchenne patients, and recommend caution and careful titration,

with the aid of a nerve stimulator, of doses of nondepolarising muscle relaxants in all patients with muscular dystrophy.

Initial reports of specific muscle biopsy findings in EDMD with type-11 fibres predominating and type-1 fibre atrophy have since been questioned.<sup>7-9</sup> The variation of muscle histology in the 10 patients reviewed by Voit *et al.*<sup>5</sup> confirmed that the clinical or genetic types of muscular dystrophy are difficult to differentiate by microscopy.<sup>5</sup> Any patient with a myopathy could develop rhabdomyolysis or malignant hyperpyrexia. It would therefore seem prudent to avoid the common trigger agents and take appropriate precautions.

Cardiac evaluation of EDMD and EDMDP patients has revealed four independent, albeit frequently combined, features:<sup>5</sup> impairment of impulse generating cells; variable sinoatrial and atrioventricular conduction defects; increased atrial and ventricular heterotopia; and functional impairment of the ventricular myocardium. It should be stressed that the cardiac involvement progresses with age and a full assessment with 24-hour ambulatory ECG is essential before sufferers are subjected to any form of anaesthesia. Cardiac pacing should be considered before general anaesthesia, in view of the fact that both atrial and atrioventricular conduction defects occur, and facilities for emergency pacing should be available whatever form of anaesthesia is undertaken. Ventricular involvement was initially thought to be a specific feature of EDMDP,<sup>14</sup> but has since been reported in the x-linked form of the disorder.<sup>5,10</sup> Cardiac depressant anaesthetic agents should be used with great caution in the presence of a ventricular myopathy and many anaesthetists consider the use of a local technique more appropriate.

Flexion of the neck, when mentioned, is always markedly restricted in EDMD patients.<sup>4,23</sup> Hypoplasia of the third to fifth cervical vertebral bodies and intervertebral discs, with fusion of the apophyseal joints, is described.<sup>15</sup> This is thought to be secondary to the immobilisation and disuse engendered by early stiffness of the posterior cervical muscles. Airway maintenance and intubation may prove difficult and careful assessment with cervical radiography should be undertaken to ascertain the degree of disability before anaesthesia is contemplated.

It can be concluded from the above that there is no ideal anaesthetic for the Emery-Dreifuss sufferer. It would seem prudent to approach each request for anaesthesia with caution. A careful evaluation before operation of the severity of the myopathy, degree of cardiac involvement and likely ease of airway maintenance should enable the risks to be gauged. Prophylactic cardiac pacing should be considered before operation. A local anaesthetic technique would, on balance, appear to be the safest option and proved uneventful in the case described. A total intravenous anaesthetic or an opioid/ventilation technique that omits suxamethonium and all trace of volatile agents would seem appropriate if a general anaesthetic cannot be avoided. Careful neuromuscular monitoring of nondepolarising muscle relaxants should be undertaken and post-operative observation in a high dependency area would seem sensible. Temperature and cardiac function should also be monitored throughout the per- and postoperative period, irrespective of the method of anaesthesia chosen, and facilities for emergency cardiac pacing should be to hand at all times.

## References

1. DREIFUSS FE, HOGAN GR. Survival in x-chromosomal muscular dystrophy. *Neurology* 1961; **11**: 734-7.
2. EMERY AEH, DREIFUSS FE. Unusual type of benign x-linked muscular dystrophy. *Journal of Neurology, Neurosurgery and Psychiatry* 1966; **29**: 338-42.
3. CESTAN R, LEJONNE V. Une myopathie avec retractions familiales. *Nouvelle Iconographie Salpêtrière* 1902; **15**: 38-52.
4. ROWLAND LP, FETELL M, OLARTE M, HAYS A, SINGH N, WANAT FE. Emery-Dreifuss muscular dystrophy. *Annals of Neurology* 1979; **5**: 111-7.
5. VOIT T, KROGMANN O, LENARD HG, NEUEN-JACOB E, WECHSLER W, GOEBEL HH, RAHLF G, LINDINGER A, NIENABER C. Emery-Dreifuss muscular dystrophy: disease spectrum and differential diagnosis. *Neuropediatrics* 1988; **19**: 62-71.
6. WATERS DD, NUTTER DO, HOPKINS LC, DORNEY ER. Cardiac features of an unusual x-linked humeroperoneal neuromuscular disease. *New England Journal of Medicine* 1975; **293**: 1017-22.
7. DICKEY RP, ZITER FA, SMITH RA. Emery-Dreifuss muscular dystrophy. *Journal of Pediatrics* 1984; **104**: 555-9.
8. HOPKINS LC, JACKSON JA, ELSAS LJ. Emery-Dreifuss humeroperoneal muscular dystrophy: an x-linked myopathy with unusual contractures and bradycardia. *Annals of Neurology* 1981; **10**: 230-7.
9. PETTY RKH, THOMAS PK, LANDON DN. Emery-Dreifuss syndrome. *Journal of Neurology* 1986; **233**: 108-14.
10. THOMAS PK, CALNE DB, ELLIOTT CF. X-linked scapuloperoneal syndrome. *Journal of Neurology, Neurosurgery and Psychiatry* 1972; **35**: 208-15.
11. HODGSON S, BOSWINKEL E, COLE C, WALKER A, DUBOWITZ V, GRANATA C, MERLINI L, BOBROW M. A linkage study of Emery-Dreifuss muscular dystrophy. *Human Genetics* 1986; **74**: 409-16.
12. THOMAS NST, WILLIAMS H, ELSAS LJ, HOPKINS LC, SARFARAZI M, HARPER PS. Localisation of the gene for Emery-Dreifuss muscular dystrophy to the distal long arm of the X chromosome. *Journal of Medical Genetics* 1986; **23**: 596-598.
13. YATES JRW, AFFARA NA, JAMIESON DM, FERGUSON-SMITH MA, HAUSMANOWA-PETRUSEWICZ I, ZAREMBA J, BORKOWSKA J, JOHNSTON AW, KELLY K. Emery-Dreifuss muscular dystrophy: localisation to Xq27.3 → qter confirmed by linkage to the factor VIII gene. *Journal of Medical Genetics* 1986; **23**: 587-90.
14. CHAKRABARTI A, PEARCE JMS. Scapuloperoneal syndrome with cardiomyopathy: report of a family with autosomal dominant inheritance and unusual features. *Journal of Neurology, Neurosurgery and Psychiatry* 1981; **44**: 1146-52.
15. MILLER RG, LAYZER RB, MELLENTHIN MA, GOLABI M, FRANCOZ RA, MALL JC. Emery-Dreifuss muscular dystrophy with autosomal dominant transmission. *Neurology* 1985; **35**: 1230-3.
16. EMERY AEH. X-linked muscular dystrophy with early contractures and cardiomyopathy (Emery-Dreifuss type). *Clinical Genetics* 1987; **32**: 360-7.
17. EMERY AEH. Emery-Dreifuss muscular dystrophy and other related disorders. *British Medical Bulletin* 1989; **45** No. 3: 772-87.
18. KATZ J, BENUMOF J, KADIS LB. *Anaesthesia and uncommon diseases: pathophysiologic and clinical correlations*, 2nd edn. Philadelphia: W.B. Saunders, 1981.
19. SMITH CL, BUSH GH. Anaesthesia and progressive muscular dystrophy. *British Journal of Anaesthesia* 1985; **57**: 1113-8.
20. BOLTSCHAUER E, STEINMANN B, MEYER A, JERUSALEM F. Anaesthesia-induced rhabdomyolysis in Duchenne muscular dystrophy. *British Journal of Anaesthesia* 1980; **52**: 559.
21. CHALKIADIS GA, BRANCH KG. Cardiac arrest after isoflurane anaesthesia in a patient with Duchenne's muscular dystrophy. *Anaesthesia* 1990; **45**: 22-25.
22. BUZZELLO W, HUTTARSCH H. Muscle relaxation in patients with Duchenne's muscular dystrophy. *British Journal of Anaesthesia* 1988; **60**: 228-31.
23. ROWLAND LP, LAYZER RB. X-linked muscular dystrophies. In: VINKEN PJ, BRUYN G, eds. *Handbook of clinical neurology*. Amsterdam: North Holland Publishing, 1979: 349-413.



CASE REPORT

## Emergency adenotonsillectomy for acute postoperative upper airway obstruction

J. R. LIVESEY, N. B. SOLOMONS AND E. A. D. GILLIES

### Summary

*Peri-operative acute upper airway obstruction may be life-threatening. A case is reported of a child with severe adenotonsillar hypertrophy who developed acute upper airway obstruction after a routine surgical procedure and required emergency adenotonsillectomy. The importance of pre-operative assessment is stressed.*

### Key words

*Ventilation; obstruction.*

*Sleep; apnoea.*

*Surgery; otolaryngological, adenotonsillectomy.*

Unrecognised and unanticipated episodes of acute upper airway obstruction may occur in association with obstructive sleep apnoea and can result in death.<sup>1</sup> This possibility should always be considered in children with obstructive sleep apnoea and in patients scheduled for adenoidectomy and tonsillectomy. A case is reported of a boy with severe upper airway obstruction from enlarged tonsils and adenoids who was awaiting adenotonsillectomy, and who underwent elective orchidopexy in the interim. Critical postoperative upper airway obstruction occurred and emergency adenotonsillectomy was necessary.

### Case history

A 4-year-old boy was admitted for orchidopexy by the urologists. He weighed 13.7 kg. He received premedication with trimeprazine (3 mg/kg) and droperidol (0.2 mg/kg), and anaesthesia was induced with nitrous oxide, oxygen and halothane. The airway became obstructed when consciousness was lost, but control was regained after insertion of a size one Guedel airway. The anaesthetist noted marked tonsillar hypertrophy. Papaveretum (3 mg) was given intravenously during the procedure. Recovery was uneventful and he was returned to the ward.

He was found to be stridulous and cyanosed 2 hours later. Administration of oxygen by mask improved his cyanosis. An urgent ENT opinion was obtained because of the rapid deterioration of his airway patency.

The clinical notes documented that he had been seen a fortnight previously in the ENT outpatient clinic. He had a long history of recurrent acute tonsillitis, mouth-breathing, snoring, rib recession and lethargy. He was a slow and noisy eater and was on the third centile for weight.<sup>2</sup> On examination he was noted to have adenoidal facies, severe tonsillar hypertrophy, cervical lymphadenopathy and bilateral secretory otitis media. His name had been placed on the 'urgent' waiting list for adenotonsillectomy, bilateral myringotomy and insertion of grommets.

The boy was pale and drowsy when seen 2 hours after his orchidopexy. He was breathing through his mouth and had marked inspiratory and expiratory stridor. He had supra-sternal and intercostal muscle retraction with palpable pulsus paradoxus. His oral cavity was dry and his tonsils were firmly adherent in the midline, causing gross obstruction of his upper airway. It was decided that he would require urgent adenotonsillectomy to relieve his obstruction. He was returned to the operating theatre and received a gaseous induction using 100% oxygen and halothane. Laryngoscopy by the anaesthetist confirmed almost total occlusion of the oropharynx by enlarged tonsils. Tracheal intubation was successful and spontaneous ventilation established with relief of his airway obstruction. An uncomplicated adenotonsillectomy and bilateral grommet insertion took place. The trachea was extubated at the end of the procedure and recovery was uneventful.

---

J.R. Livesey, BA, FRCS(Ed), ENT Senior House Officer, N.B. Solomons, FRCS, ENT Senior Registrar, E.A.D. Gillies, MB, ChB, DA, Anaesthetic Senior House Officer, The Royal Surrey County Hospital, Egerton Road, Guildford, Surrey GU2 5XX.

Accepted 27 February 1990.

The snoring and sleep apnoea ceased that night. He was very active around the ward after eating a large breakfast the next day and had no signs of airway obstruction. He was discharged on the third postoperative day. His mother commented on the immediate improvement in his general well-being at a follow-up visit in the outpatient clinic. He was sleeping all night without snoring, had a markedly increased appetite and was a much healthier and happier child.

### Discussion

The most likely explanations for the sudden onset of acute airway obstruction in this child are that the oropharynx was dry as a result of pre-operative starvation and the use of unhumidified anaesthetic gases, and that there may have been a degree of sedation remaining from the effects of premedicant and anaesthetic drugs. In addition, there may have been prolonged periods of mouth breathing in the postoperative period, causing further drying of the oropharyngeal mucosa. It is possible that the Guedel airway may have produced minor trauma to the tonsils.

The frequency of adenotonsillectomy for recurrent pharyngotonsillitis has fallen dramatically in the decades since the introduction of oral antibiotics.<sup>3</sup> Thus there is probably a relatively increased incidence of airway obstruction secondary to prolonged adenotonsillar hypertrophy. It is only in recent years that there has been an increased awareness of tonsillar enlargement as a cause of airway obstruction, and of the dangers associated with such obstruction.<sup>4</sup> The vast majority of adenotonsillectomies performed at the Children's Hospital of Philadelphia are now done for chronic airway obstruction rather than recurrent pharyngotonsillitis.<sup>5</sup>

There is a spectrum of upper airway obstruction which ranges from mild and occasional snoring through to the full-blown sleep apnoea syndrome. Children with adenotonsillar hypertrophy may have symptoms anywhere within this spectrum. The definition of sleep apnoea is the cessation of airflow at the level of the nostrils and mouth lasting at least 10 seconds, whilst the sleep apnoea syndrome comprises at least 30 apnoeic episodes over 7 hours of nocturnal sleep, during both rapid and nonrapid eye movement (NREM) sleep, and with some apnoeas in a repetitive sequence in NREM sleep.<sup>6</sup> The use of polysomnography has helped in the diagnosis and quantification of upper airway obstruction.<sup>3</sup> Adenotonsillectomy provides immediate relief of upper airway obstruction in these children.<sup>4</sup>

Additional features in children with upper airway obstruction include nocturnal enuresis,<sup>7</sup> daytime somnolence and irritability, anosmia, orthodontic malformations<sup>5</sup> and pectus excavatum.<sup>8</sup> The cardiopulmonary consequences of longstanding upper airway obstruction include arrhythmias, right atrial and ventricular hypertrophy, congestive heart failure and cor pulmonale, which occur as a result of pulmonary hypertension.<sup>8,9</sup> The mechanism is thought to be secondary to alveolar hypoxia which in turn leads to pulmonary vasoconstriction.<sup>10</sup> These severe sequelae normally result only from longstanding obstruction, and are fortunately uncommon.<sup>4</sup> Reversal of several of the sequelae has been reported following adenotonsillectomy.<sup>4,9,10</sup>

Other conditions associated with snoring and apnoea include: craniofacial anomalies (e.g. achondroplasia and

Treacher Collins syndrome) where normal lymphoid tissue volume in a small airway may produce problems; Hunter's and Hurler's syndromes in which complicated airway problems often include adenotonsillar obstruction; Down's syndrome, in which snoring and moderate to severe obstruction are common; stable or progressive neuromuscular disorders, when lack of pharyngeal support may allow collapse of the tonsils and adenoids into the airway;<sup>4</sup> glottic obstruction, including vocal cord paralysis,<sup>11</sup> and subglottic obstruction.

The history is of great importance in assessment of the risk of serious upper airway obstruction. Examination may be corroborative, particularly for tonsillar hypertrophy. A lateral soft tissue cervical X ray may show a reduction of the nasopharyngeal airway from adenoidal hypertrophy.<sup>3</sup>

Parents of small children are not always present when the anaesthetist makes his pre-operative assessment and it is helpful if the surgeon informs the anaesthetist pre-operatively if the patient is prone to upper airway obstruction or has sleep apnoea. The choice of premedication is important. Sedatives and antisialogogues may have adverse effects on an already compromised airway. Opioids administered in the postoperative period increase the risk of apnoeic episodes. We believe that in patients with symptomatic adenotonsillar hypertrophy who present for other types of surgery it is advantageous to perform adenotonsillectomy at the same operation.

In conclusion, the recognition of hypertrophy of the tonsils or adenoid and obstructive sleep apnoea as causes of peri-operative acute upper airway obstruction and their skilled management should minimise the morbidity and mortality of this potentially life-threatening disorder.

### References

1. KRAVATH RE, POLLAK CP, BOROWIECKI B, WEITZMAN ED. Obstructive sleep apnea and death associated with surgical correction of velopharyngeal incompetence. *Journal of Pediatrics* 1980; **96**: 645-8.
2. TANNER JM, WHITEHOUSE RH. *Boys: birth-19 years height and weight*. 1983 Ware, Heris. Castlemead for Ward's Publishing Services.
3. GRUNDFAST KM, WITTICH DJ. Adenotonsillar hypertrophy and upper airway obstruction in evolutionary perspective. *Laryngoscope* 1982; **92**: 650-5.
4. GUILLEMINAULT C, ELDRIDGE FL, SIMMONS FB, DEMENT WC. Sleep apnea in eight children. *Pediatrics* 1976; **58**: 23-30.
5. POTSIC WP. Tonsillectomy and adenoidectomy. *International Anesthesiology Clinics* 1988; **26**: 58-60.
6. GUILLEMINAULT C, TILKIAN A, DEMENT WC. The sleep apnoea syndromes. *Annual Review of Medicine* 1976; **27**: 465-84.
7. WEIDER DJ, HAURI PJ. Nocturnal enuresis in children with upper airway obstruction. *International Journal of Pediatric Otorhinolaryngology* 1985; **9**: 173-82.
8. RICHTER I, MARTINS M. Occlusive adenoids causing cor pulmonale and pectus excavatum in a child. *Monatsschrift Kinderheilkunde* 1981; **129**: 175-7.
9. LEVY AM, TABAKIN BS, HANSON JS, NARKEWICZ RM. Hypertrophied adenoids causing pulmonary hypertension and severe congestive heart failure. *New England Journal of Medicine* 1976; **277**: 506-11.
10. MASSUMI RA, SARIN RK, POOYA M, REICHELDERFER TR, FRAGE JR, RIOS JR, AYESTERIAN E. Tonsillar hypertrophy, airway obstruction, alveolar hypoventilation, and cor pulmonale in twin brothers. *Diseases of the Chest* 1969; **55**: 110-4.
11. WESTERMAN DE, FERGUSON AD. Obstructive sleep-induced apnoea due to bilateral recurrent laryngeal nerve palsies. *South African Medical Journal* 1978; **54**: 825-7.

## Non-drug related asystole associated with anaesthetic induction

T. K. KEANE, P. J. HENNIS AND M. TH. E. BINK-BOELKENS

### Summary

*A patient is presented where routine venepuncture associated with anaesthetic induction resulted in bradycardia and asystole. The case highlights the need for special caution with, and ECG monitoring throughout induction for, patients with a history of syncope. It also demonstrates the need for caution when attributing cardiovascular events during induction to the effect of the induction agents used.*

### Key words

Anaesthesia; intravenous.

Complications; asystole.

Arrhythmias associated with the effect of anaesthetics or other medication on the cardiac conducting tissues are often reported. It is unusual to find a patient where the mildly painful procedures associated with an intravenous induction result in a profound bradycardia and cardiac arrest. This report describes such a patient.

### Case History

A 20-year-old male patient was scheduled for a routine follow-up cardiac catheterisation. He had had a total correction of a Fallot's tetralogy in 1976, and the operation had achieved a good result, with slight residual pulmonary stenosis. His general health and exercise tolerance were good, and he had been receiving no medication since this operation. His pre-procedure ECG showed a high-normal P-R interval (0.2 second) with no other abnormality.

The procedure was commenced in the usual manner. ECG monitoring was applied and lignocaine 1% was infiltrated subcutaneously over the femoral area. The patient had a short syncopal episode when the introducing needle was inserted into this area, with a heart rate of 20/minute. This spontaneously recovered in 20 seconds and the procedure was continued. A similar sinus bradycardia with a heart rate of 20/minute occurred when the venous sheath was inserted, with recovery taking 40 seconds. It was decided to postpone the catheterisation and do it later under general anaesthesia. The patient had not complained of any painful sensation during this attempted procedure,

and a postoperative ECG showed no change from that before the procedure.

The patient was premedicated with oral diazepam 15 mg before the second catheterisation attempt and appeared adequately sedated. However, when a 22-gauge cannula was inserted into the dorsum of his hand he developed bradycardia. This rapidly progressed to asystole, which was associated with a tonic-clonic convulsion. Oxygen 100% was given and cardiac massage was performed. A bolus dose of intravenous atropine 1 mg resulted in a sinus rate of 80/minute. The procedure was again abandoned. A subsequent detailed family history revealed that two of the patient's immediate family, his mother and twin brother, had experienced similar syncopal attacks under stressful circumstances, but had not had a documented cardiac arrest, nor any history of hospital treatment. A repeat ECG was identical to the pre-arrest one. A 24-hour Holter recording showed occasional multifocal ventricular ectopics with a short period of nocturnal Wenckebach A-V heart block with a maximal pause of 2.9 seconds. Such changes alone would not explain these attacks and a neurogenic basis was thought to be responsible. The cardiologist decided that a pacemaker should be inserted to prevent further episodes of cardiac arrest, but because of his reaction to painful stimuli the procedure had to be carried out under a general anaesthetic.

The patient was premedicated with oral nitrazepam 10 mg on the night before operation and with oral lorazepam 2.5 mg, and oral atropine 2 mg, 1.5 hours before

T.K. Keane,\* MB, BCh, FFARCS, Cardiac Anaesthetic Fellow, P.J. Hennis, MD, PhD, Professor of Anaesthesia, Department of Anaesthesia, M.Th.E. Bink-Boelkens, MD, PhD, Staff Member, Division of Paediatric Cardiology, Department of Paediatrics, Academisch Ziekenhuis Groningen, Oostersingel 59, 9713 EZ Groningen, The Netherlands.

\*Present address: Department of Anaesthesia, University Hospital, Clifton Boulevard, Nottingham NG1 2UH.

Accepted 14 March 1990.

operation. Oral atropine 2 mg was chosen since the subcutaneous or intravenous routes could elicit bradycardia or cardiac arrest. A local anaesthetic cream was applied to the dorsa of both hands. Monitoring was commenced on arrival in the operating theatre; the pre-induction arterial blood pressure and heart rate were 120/60 mmHg and 80/minute respectively. A gaseous induction with 33% oxygen, 66% nitrous oxide and halothane up to 4% was started. An intravenous cannula was inserted uneventfully, and pancuronium 6 mg was given and the trachea intubated. Anaesthesia was maintained with nitrous oxide (66%) oxygen (33%) and halothane (1%). Two bolus doses of intravenous alfentanil 0.5 mg each were given. A bipolar pacing electrode was inserted via the subclavian vein and a site with a suitable threshold easily found in the right ventricle. A VVI pacemaker was implanted and a rate of 65/minute was selected, since it was lower than his normal sinus rate but expected to be high enough to provide haemodynamic stability during recovery from anaesthesia. It was considered safe to reverse the muscle relaxant with neostigmine and methyl-atropine 2.5 mg and 0.5 mg respectively since a functional pacemaker was *in situ*.

The dressing and drain were removed on the first post-operative day, and the ECG was recorded during the procedure. The patient's pre-procedure sinus rate was 70/minute, which decreased to less than the preset pacemaker rate for 12 minutes when the drains were removed. The patient became sweaty and pale but remained conscious. The pacemaker rate was lowered to 45/minute after recovery of his sinus rate to its previous level.

A challenge was applied on the fourth day after operation. A 20-gauge cannula was inserted into the dorsum of his left hand, which resulted in a bradycardia of less than 45/minute (the pacemaker preset rate) after a latent period of 48 seconds. The patient became sweaty, pale and felt dizzy, but again remained conscious. The blood pressure was 85/45 mmHg, and increased to 100/65 mmHg after 30 seconds. The sinus rate increased to 60/minute after 2.5 minutes of pacing and the blood pressure increased to 120/60 mmHg. These haemodynamic variables during an attack indicated that a setting of 45/minute was adequate to prevent future attacks.

### Discussion

This case is reported to draw attention to the existence of a small number of the population who have severe vasovagal reactions to painful and distressing events, occasionally resulting in asystole.<sup>1</sup> It is easy to underestimate the significance of a history of simple 'faints' in an otherwise fit, healthy patient, who has no apparent ECG abnormality. Profound cardiovascular consequences may follow if such a history is ignored. This case also highlights the need for caution in interpreting the causes of bradycardia and

cardiovascular collapse during induction and particularly the role of specific anaesthetic agents.<sup>2,3</sup> The period that elapsed between venepuncture and the onset of asystole would have allowed time for the administration of an induction agent during most routine procedures. The subsequent cardiovascular collapse would then have been interpreted as an adverse effect of this agent. This caution is especially needed when a history of such episodes is present.<sup>2</sup>

The need for pacemaker insertion in a population subgroup who have syncopal attacks without any cardiac abnormality likely to cause these attacks is well documented.<sup>4,6</sup> These episodes are usually attributed to neurally mediated mechanisms. Firm diagnosis is often difficult, since these episodes are not always reproducible in front of medical witnesses. Various tests have been used to assess susceptibility to neurogenic syncope, including upright tilt testing and isoprenaline infusion.<sup>5</sup> The tests were not required in this case because of the two documented episodes. The literature also indicates that the incidence of recurrence of syncope is high and may result in sudden death.<sup>6</sup> This patient's tendency to syncope was most probably neurogenic in origin, but may have been exacerbated by his previous correction of Fallot's tetralogy.

Our anaesthetic approach was not unusual. Atropine was chosen to lessen the risk of inadvertent syncope before and during induction and an intravenous approach to induction was considered unwise, so halothane was selected to provide a smooth rapid induction. These properties outweighed its potential for arrhythmia production. Pancuronium was chosen for its chronotropic effect and because of the estimated duration of the procedure (1.5 hours). Reversal with neostigmine was considered safe after pacemaker insertion. In view of the strong positive result of the challenge, it is clear that our concern about induction technique was justified.

### References

1. SCHRAEDER PL, PONTZER R, ENGEL TR. A case of being scared to death. *Archives of Internal Medicine* 1983; **143**: 1793-4.
2. DORRINGTON KL. Asystole with convulsion following a subanaesthetic dose of propofol plus fentanyl. *Anaesthesia* 1989; **44**: 658-9.
3. RUIZ K. Asystole on induction. *Anaesthesia* 1989; **44**: 698-9.
4. RATTES MF, KLEIN GJ, SHARMA AD, BOONE JA, KERR C, MILSTEIN S. Efficacy of empirical cardiac pacing in syncope of unknown cause. *Canadian Medical Association Journal* 1989; **140**: 381-5.
5. ALMOUST A, GOLDENBURG IF, MILSTEIN S, CHEN MY, CHEN XC, HANSEN R, GORNIC CC, BENDITT DG. Provocation of bradycardia and hypotension by isoproterenol and upright posture in patients with unexplained syncope. *New England Journal of Medicine* 1989; **320**: 346-51.
6. RAVIELE A, PROCLEMER A, GASPARINI G, DI-PEDE F, DELISE P, PICCOLO E, FERUGLIO GA. Long-term follow-up of patients with unexplained syncope and negative electro-physiological study. *European Heart Journal* 1989; **10**: 127-32.

## Rupture of the oesophagus during cricoid pressure

S. J. RALPH AND C. A. WAREHAM

### Summary

*Rupture of the oesophagus occurred during the application of cricoid pressure at induction of anaesthesia when the patient vomited. The patient, who was bleeding from a gastric ulcer, was found to have a lower oesophageal tear which, although repaired at operation, resulted in a fatal mediastinitis.*

### Key words

*Complications; oesophageal rupture.  
Larynx; cricoid pressure.*

### Case history

An 81-year-old female was admitted for investigation of upper abdominal pain and vomiting. Oesophago-gastroscopy revealed a large malignant-looking gastric ulcer in the region of the pylorus. The oesophagus was mildly inflamed but otherwise normal, and partial gastrectomy planned. She was in good health apart from quite severe and disabling rheumatoid arthritis and taking no medication.

The day before surgery she had a brisk haematemesis which required resuscitation with intravenous fluid including blood. She became haemodynamically stable and no immediate intervention was planned. She had a second haematemesis 6 hours later which again required resuscitation, and it was decided to operate.

She was transferred to the operating theatre for laparotomy after further resuscitation. Rapid sequence induction, with cricoid pressure and suxamethonium was planned. Monitoring was started and she was given 100% oxygen through a mask for 5 minutes. Cricoid pressure was applied at the start of induction anaesthesia with etomidate. She actively vomited without warning immediately after 12 mg etomidate had been given but before loss of consciousness. Cricoid pressure was discontinued, she was turned on to her left side and the pharynx aspirated. Suxamethonium was given and the trachea intubated with a cuffed tracheal tube.

At operation a large actively bleeding ulcer in the region of the pylorus was oversewn. The surgeon also noticed that the gastric cardia was bruised and swollen with a 10 cm

longitudinal split in the lower oesophagus which extended across a small hiatus hernia. The split was complete through the oesophageal wall. The rupture was repaired, partial gastrectomy with retrocolic anastomosis performed and the patient transferred to the intensive care unit. She developed mediastinitis, respiratory and renal failure and died 10 days after operation.

### Discussion

Cricoid pressure was described by Sellick in 1961<sup>1</sup> when he showed that firm pressure on the cricoid cartilage with the neck in extension compresses the oesophagus and prevents the regurgitation of gastric contents into the pharynx. Subsequently it was shown that a force of 44 N is required to occlude the oesophagus.<sup>2</sup> It has also been shown that cricoid pressure is effective with a nasogastric tube *in situ*.<sup>3</sup> A rapid sequence induction is recommended for patients at risk of inhalation of gastric contents, with cricoid pressure applied before the start of intravenous induction.<sup>4–6</sup> Most patients find this tolerable although it is uncomfortable.

Oesophageal rupture has always been considered a risk if active vomiting occurs when cricoid pressure is applied, although it has never been described. Spontaneous rupture of the oesophagus can occur in association with active vomiting when the cricopharyngeus muscle fails to relax during vomiting. Pressure in the oesophagus increases so rapidly that rupture occurs at the weakest point of the oesophagus, usually on the posterior wall at the extreme lower end.<sup>7</sup> Symptoms are usually severe and dramatic in onset. They include chest or abdominal pain, vomiting and

S.J. Ralph, BMedSci, BM, BS, FFARCS, Senior Registrar, C.A. Wareham, BM, DRCOG, Senior House Officer, Department of Anaesthetics, Royal Hallamshire Hospital, Sheffield S10 2RX.

Accepted 1 April 1990.



dyspnoea. Signs are also immediate and include tachycardia, tachypnoea, hypotension and fever. Surgical emphysema may also occur.<sup>7,8</sup>

Spontaneous rupture is an unlikely diagnosis in this patient. The oesophagus was seen to be normal on gastroscopy and she had not been vomiting profusely. In addition, she did not display the dramatic symptoms and signs described above. It is likely that the perforation occurred when she vomited during induction of anaesthesia with cricoid pressure applied. Presumably the mechanism is similar to that of spontaneous rupture with cricoid pressure replacing a tonic cricopharyngeus muscle. The hiatus hernia provided the weak point and the focus for the rupture.

The correct timing of the application of cricoid pressure is important. It is recommended that application should be before induction of anaesthesia, and if the patient then vomits, as in this case, with full muscle power, there is high risk of oesophageal rupture. Sellick advocates that the onset of unconsciousness, the achievement of full muscle relaxation and application of full cricoid pressure should occur simultaneously.<sup>9</sup> In this way cricoid pressure prevents regurgitation and a paralysed patient cannot vomit, but in elderly poor risk patients, with slow circulation time, this ideal may be difficult to achieve. We would recommend Sellick's description of light application of cricoid pressure before induction which is increased to reach maximum force as the patient loses consciousness. Cricoid pressure should be released immediately if active vomiting occurs.

We believe this to be the first described case of rupture of

the oesophagus consequent to active vomiting with cricoid pressure applied. Active vomiting should be considered a contraindication to cricoid pressure together with laryngeal trauma, cervical spine injury to the region of the cricoid, cervical arthritis and immobile neck.<sup>6</sup> In all other cases the benefits of cricoid pressure as demonstrated by 30 years of clinical practice, far outweigh the risk of this rare complication.

### References

1. SELICK BA. Cricoid pressure to control regurgitation of stomach contents during induction of anaesthesia. *Lancet* 1961; ii: 404-6.
2. WRAIGHT WJ, CHAMNEY AR, HOWELLS TH. the determination of an effective cricoid pressure. *Anaesthesia* 1983; 38: 461-6.
3. SELICK BA. The prevention of regurgitation during induction of anaesthesia. *Proceedings of the First Congress of Anaesthesiology, Vienna 1962*; 1: 89.
4. GREGORY IC. Anaesthesia and the gastro-intestinal tract. In: CHURCHILL-DAVIDSON HD, ed. *A Practice of Anaesthesia*, 5th edn. London: Lloyd-Luke, 1984: 939-53.
5. MORGAN M. Obstetric Anaesthesia and Analgesia. In: NIMMO WS, SMITH G, eds. *Anaesthesia*, Vol. 1. Oxford: Blackwell, 1989: 691-715.
6. CROWLEY DS, GIESECKE AH. Bimanual cricoid pressure. *Anaesthesia*, in the press.
7. The oesophagus. In: HARDING-RAINS AJ, MANN CV, eds. *Bailey and Love's Short Practice of Surgery*, 20th edn. London: H. K. Lewis & Co Ltd, 1988: 827-54.
8. MOOSA AR, WINANS CS. Benign disorders of the oesophagus and diaphragm. In: CUSCHIERI A, GILES GR, MOOSA AR, eds. *Essential surgical practice*. Bristol: Wright, 1982: 597-622.
9. SELICK BA. Rupture of the oesophagus following cricoid pressure? *Anaesthesia* 1982; 37: 213-14.

## Accidental bronchial intubation with RAE tubes

A. E. BLACK AND A. M. MACKERSIE

### Summary

*Preformed tracheal tubes are used frequently in paediatric anaesthesia. A feature which contributes to their popularity is the belief that they can be positioned more reliably than conventional tracheal tubes because of their design. We studied a group of 40 patients in whom the incidence of bronchial intubation was 20%. The tube was too long in 32% of patients, although the tube size was appropriate for the child's age in all patients. The consequences and outcome of this complication are discussed.*

### Key words

*Intubation, tracheal; complications.*

*Equipment; RAE tracheal tubes.*

The use of preformed Ring, Adair, Elwyn pattern tubes (RAE, Mallinckrodt) in children is becoming increasingly common in the belief that positioning is achieved predictably.<sup>1</sup> However, clinical assessment of correct position is known to be unreliable. The identification of an episode of bronchial intubation with a RAE tube that resulted in arterial desaturation, detected by pulse oximetry, prompted us to investigate the position of RAE tubes in children during myelography.

### Methods

The study group consisted of 50 consecutive patients who underwent myelography with the use of general anaesthesia. The study was retrospective so that current practice would be reviewed. The case notes and anaesthetic records of all the children were studied. All patients were in prone position, tilted appropriately (depending on the level to be studied) and the neck was partly extended, during myelography. RAE tubes were used routinely and their position was assessed clinically. X rays taken during the myelograms were reviewed and the position of the distal tip of the tracheal tube identified. The level of the tube in relation to the carina was measured, and the position of the carina was related to the appropriate thoracic vertebral level.

### Results

Full records and adequate X rays were found in 40 patients, but the X rays were uninformative in the remainder either because there were no X rays of the chest or the radiopaque dye obscured identification of the tube or carina.

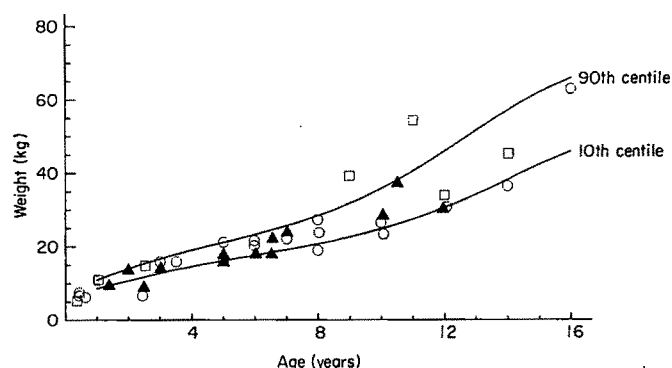


Fig. 1. Age:weight distribution of children. Tracheal tube positions: ○, good; ▲, tube too long; □, tube too short.

The age range was 3 months to 16 years (median 6 years). Weights ranged from 5.3 to 62 kg. The great majority were between the 10th and 90th centile for weight to age distribution (Fig. 1). The indications for myelography are summarised in Table 1.

Table 1. Demographic data.

Males:females	26:14
Age; years	0.25–16 (median 6)
Weight; kg	5.3–62
Diagnoses	
Cerebral tumour	9
Multiple congenital abnormality	4
Lumbosacral abnormality	13
Other (scoliosis, abnormal gait, abdominal pain, etc.)	14

A.E. Black, FFARCS, Registrar, A.M. Mackersie, FFARCS, Consultant, Department of Anaesthesia, Hospital for Sick Children, Great Ormond Street, London WC1N 3JH

Accepted 15 May 1990.

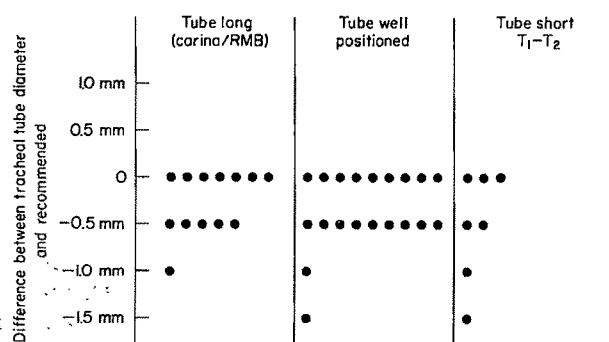


Fig. 2. Relationship between the position of the tip of the tracheal tube and the difference between internal diameter of the tube used and the appropriate diameter calculated from the patient's age. RMB; right main bronchus.

The carina was clearly identified between T<sub>4</sub> and T<sub>5</sub> in 92.5% of the patients. The tracheal tube was at the carina in five children and in the right main bronchus in eight. Thus, the tube was too long in 32% (95% confidence intervals 18.6–49.1% of children). Bronchial intubation had occurred in 20% (95% confidence intervals 9.1–35.7%).

The internal diameter of the RAE tube was either the size that would have been anticipated from the standard formula, i.e.  $((\text{age}/4) + 4)$  mm, or 0.5–1.0 mm smaller (Fig. 2) in all the children in whom the tube was too long. There was no child in whom the tube was larger than predicted for its age.<sup>2</sup> The manufacturers of RAE tubes recommend a larger internal diameter tube than that predicted by the standard formula (Table 2).

### Discussion

The investigation of the position of the tip of the tracheal tube in children who underwent myelography allowed the distance of the patient from the X ray film to be standardised so that the magnification factor remained constant. Consequently, the position of the tube and its relationship to the carina can be interpreted meaningfully. The ideal position of the tube is in midtrachea and in a recent study of elective tracheal intubation using pre-measured plain nasal tubes only 1.6% were too long.<sup>2</sup> In our study, there was a high incidence (32.5%) of intubations in which the tube was too long when preformed oral tubes were employed. Movement or repositioning of the patient during anaesthesia may affect the position of the tube, as does normal ventilation.<sup>3</sup> In these circumstances the tip may be advanced into a bronchus if it already lies in the lower trachea. Such an event may not be identified easily during operation.

It was interesting that in our study the carina was identified easily at T<sub>4</sub>–T<sub>5</sub> in 92.5% of the group and that in

Table 2. Ages suggested by the manufacturer for each tube diameter compared with those derived from formula.<sup>2</sup>

Internal diameter in mm	Age of child by formula $((\text{age}/4) + 4)$ mm	Age of child from data on RAE tube
3.0		0–4 weeks
3.5		1–6 months
4.0		0.5–1 year
4.5	2	1–2 years
5.0	4	2–4 years
5.5	6	4–5.5 years
6.0	8	5.5–7.5 years
6.5	10	7.5–9 years
7.0		> 9 years

seven patients the tip of the tube lay at T<sub>1</sub>–T<sub>2</sub>; this indicates that there was a risk of accidental extubation in these patients. There was no morbidity despite a 20% incidence of bronchial intubation. It is probable that the presence of the Murphy's eye allows some ventilation of the left lung during episodes of bronchial intubation,<sup>1</sup> but adequate ventilation cannot be assured.

Presence of this eye may also account for the anaesthetists' clinical assessment that the tube was positioned correctly immediately after its insertion. The use of anatomically preformed tubes is likely to increase with the introduction of preformed 'north-facing' tracheal tubes. We believe that it is important to be aware of the high risk of bronchial intubation. The routine use of pulse oximetry may help to identify bronchial intubation if saturation does decrease, although this may not occur if a high inspired oxygen fraction is employed and episodes of under-ventilation may go unobserved.

There are many advantages of RAE tubes, but the concept that bronchial intubation is unlikely and that the acute angle of the tube, with its spring-like resilience,<sup>1</sup> in some way keeps it in the correct position has been disproved. A tube which touches the carina may cause coughing and irregular respiration, and it may advance to cause trauma and oedema to the small bronchus. It should be recognised that bronchial intubation is a common event and that the age recommendations on the side of the tube can be misleading. Routine use of pulse oximetry is indicated.

### References

1. RING WH, ADAIR JC, ELWYN RA. A new pediatric endotracheal tube. *Anesthesia and Analgesia* 1975; **54**: 273–4.
2. YATES AP, HARRIES AJ, HATCH DJ. Estimation of nasotracheal tube length in infants and children. *British Journal of Anaesthesia* 1987; **59**: 524–6.
3. KUHN LS, POZNANSKI AK. Endotracheal tube position in the infant. *Journal of Pediatrics* 1971; **78**, 991–6.

## A comparison of patient rewarming devices after cardiac surgery

M. M. IMRIE, M. WARD AND G. M. HALL

### Summary

*Three regimens for rewarming patients after cardiac surgery involving hypothermic cardiopulmonary bypass were studied in 30 patients. The control group (n = 10) received no active rewarming, the oesophageal group (n = 10) was warmed centrally using an oesophageal heat exchanger and the radiant group (n = 10) was warmed peripherally with an overhead radiant heater. There were no statistically significant differences between the groups apart from the higher mean skin temperatures in the peripheral group.*

### Key words

*Temperature; body.  
Surgery; cardiovascular.  
Equipment.*

A device for noninvasive central rewarming of hypothermic patients by recirculation of warm water through an oesophageal tube has recently become available (The Exacon thermal therapy system) and was effective in experimental hypothermia in dogs,<sup>1</sup> accidental hypothermia in humans<sup>2</sup> and in prevention of perioperative hypothermia.<sup>3</sup> There have been no studies using this technique after cardiopulmonary bypass procedures; however, peripheral warming using a radiant overhead heater was demonstrated to be effective in this situation.<sup>4–6</sup> This study was designed to compare the efficacy of central oesophageal rewarming with peripheral rewarming using a radiant overhead heater (the Aragona mobile thermal ceiling) after cardiac surgery.

### Methods

Thirty patients after routine coronary artery bypass grafting were randomly allocated to one of three groups: a control group, no active rewarming in the postoperative period; an oesophageal group, rewarming using the Exacon TT8200 thermal therapy system. This consists of a disposable double lumen oesophageal tube and a base unit comprising water heater, circulating pump and monitor/ alarm module. Sterile distilled water is circulated through the oesophageal tube at a rate of 3 litres/minute. The temperature of the circulating water is variable and in this study the maximum temperature of 42°C was used. The oesophageal tube was inserted in theatre after heparinisation had been reversed; a radiant group, rewarming using

the Aragona mobile thermal ceiling. The heating surface was mounted approximately 1 m above the patients and the output adjusted initially to maximum but then decreased to maintain skin temperature at 37°C. The patients were treated identically in all other respects.

Anaesthesia consisted of premedication with papaveretum, hyoscine and droperidol given 1–2 hours pre-operatively and induction with a sleep dose of thiopentone and fentanyl (50 µg/kg). Neuromuscular blockade was achieved using pancuronium (0.15 mg/kg) and anaesthesia was maintained with 50% nitrous oxide in oxygen; muscle relaxation was not reversed at the end of surgery. Bypass temperatures of 27–28°C were used for all patients. Temperature monitoring probes were attached on arrival in the intensive care unit (ICU) and allowed to stabilise for 15 minutes before the readings started. Intermittent positive pressure ventilation was continued after operation with oxygen-enriched air. Humidification of inspired gases was achieved using the Engstrom Edith heat and moisture exchanger. Postoperative sedation and pain relief were provided using intermittent intravenous boluses of midazolam and papaveretum. Vasodilators (sodium nitropruside or glyceryl trinitrate) and inotropes (dopamine) were administered by intravenous infusion as required to control systolic blood pressure. All patients were nursed covered to the waist with an aluminised reflecting blanket.

The following demographic and clinical data were recorded for all patients: weight, sex, age, current medication, bypass duration, and time from termination of bypass

M.M. Imrie, MB, BS, BSc, FFARCS, Senior Registrar, G.M. Hall, MB, BS, PhD, FI Biol, FFARCS, Professor of Clinical Anaesthesia, The Royal Postgraduate Medical School, Hammersmith Hospital, Du Cane Road, London W12 0HS, M. Ward, MB, BS, FCAnaes, Registrar, Edgeware General Hospital, Edgeware, Middlesex HA8 0AD.

Accepted 22nd June 1990

to arrival in ICU. Ambient, tympanic membrane and skin temperatures were measured every 15 minutes during the study period. Skin temperature was measured at the following sites: anterior surface of chest above nipple, lateral surface of midpoint of upper arm, anterior surface of midpoint of thigh, lateral surface of midpoint of calf.

Ellab (Electrolaboriet, Copenhagen) thermocouples and recording equipment were used for all temperature measurements and the equipment was calibrated against a National Physical Laboratory mercury in glass thermometer before the study started. Skin probes were attached using adhesive orthopaedic felt to provide insulation from external heat sources.

Cardiovascular variables, systolic arterial pressure, diastolic arterial pressure, mean arterial pressure, mean central venous pressure and heart rate were also measured every 15 minutes.

**Shivering.** Shivering and muscle tone were assessed using the method of Joachimsson.<sup>5</sup> This method grades both shivering and muscle tone as 0, none; 1, moderate; 2, severe. The two scores were combined to give a shivering score with five levels, 0–4.

**Drugs.** The doses of all sedative or analgesic drugs and the infusion rates of vasodilators and inotropes were recorded.

Rewarming in groups 2 and 3 started once the first set of readings had been made on the intensive care unit and continued until the tympanic membrane temperature reached 37°C. Readings continued for 2 hours after rewarming was stopped.

**Calculations.** Mean skin temperature ( $T_{\text{skin}}$ ) was calculated according to the method of Ramanathan<sup>7</sup> and mean body temperature ( $T_{\text{body}}$ ) derived using:  $T_{\text{body}} = (0.66 \times T_{\text{core}}) + (0.34 \times T_{\text{skin}})$ . Total body heat (TBH) was calculated as from this:  $\text{TBH} = T_{\text{body}} \times \text{body weight (kg)} \times 3.475 \text{ kJ}$

where 3.475 kJ/°C. (0.83 Cal/°C.) is the specific heat of the human body.<sup>8,9</sup>

**Data analysis.** Data were analysed using Statview 512 on the Apple Macintosh. One- and two-way analysis of variance (ANOVA) were used for within-group and between-group differences. Sheffes test of significance was used where ANOVA detected a difference.

## Results

All data are given as mean values (standard deviation). The patient characteristics for the three groups are shown in Table 1. There were no significant differences between

**Table 1.** Patient characteristics. Values are expressed as mean (SD).

	Group 1 (Control)	Group 2 (Oesophageal)	Group 3 (Radiant)
Number of patients	10	10	10
Gender; male/female	8/2	9/1	10/0
Age; years	57.4 (7.5)	54.6 (7.9)	57 (9.5)
Weight; kg	78.8 (13.6)	75.9 (10.4)	77.1 (13.3)
Duration of bypass; minutes	94.5 (20.2)	98.5 (18.4)	100.5 (22.6)
Time after bypass; minutes*	84.3 (21.9)	78.2 (12.8)	82.1 (12.4)

\*Time after bypass is the time from the end of bypass to arrival in the intensive care unit.

There were no significant differences between groups.

**Table 2.** Rewarming data.

	Group 1 (Control)	Group 2 (Oesophageal)	Group 3 (Radiant)
Number of patients	10	10	10
Ambient temperature; °C	25.3 (1.2)	25.2 (1.2)	25.2 (0.8)
Rate of rise of $T_{\text{core}}$ ; °C/hour	1.0 (0.3)	1.4 (0.6)	1.1 (0.3)
Rate of rise of $T_{\text{skin}}$ ; °C/hour	1.2 (0.2)	1.3 (0.5)	1.9 (0.4)
Rate of rise of TBH; kJ/hour	303 (78)	361 (148)	355 (94)
Total shivering score (arbitrary units)	8.5 (5.7)	10.2 (11.7)	4.1 (4.1)

Data given as; Mean value (standard deviation).

No significant differences between groups.

the two groups with regard to any of the patient variables measured before the start of the study.

**Drugs.** The three groups were comparable regarding pre-operative and peroperative medication and postoperative requirements for sedative and vasodilator agents.

**Cardiovascular data.** There were no significant differences in arterial pressures, central venous pressure and heart rate between the groups at any time throughout the study period.

**Rewarming data.** The ambient temperatures during the study period are shown in Table 2. The rate of change of tympanic membrane temperature during the rewarming period was calculated as;

$$\frac{\text{First temperature} > 37^{\circ}\text{C} - \text{Temperature on arrival on ICU}}{\text{time taken for temperature increase}} \text{ } ^{\circ}\text{C/hour}$$

The rates of change of mean skin temperature and total body heat were similarly calculated over the same time interval (Table 2).

There were no differences in the tympanic membrane temperatures between the groups at any time during the study period (Fig. 1). The slightly more rapid increase in tympanic membrane temperature in the oesophageal group failed to reach statistical significance (Table 2). The mean skin temperature in the radiant group was significantly greater than the oesophageal group at 45 minutes (radiant group 32.1(0.7)°C, oesophageal group 30.9(1.2)°C.  $p < 0.05$ ) and the control group at 1 hour 15 minutes (radiant group 33.3(0.7)°C, control 32.1(0.8)°C.  $p < 0.05$ ). These differences were maintained for the remainder of the study period. There were no differences in total body heat or change of total body heat between the groups at any time during the period of the study (Fig. 1).

To examine the postwarming period the temperature data were aligned around the first temperature greater than 37°C and data points for the next 2 hours compared. The tympanic membrane temperatures of all groups continued to increase in the postwarming period and there were no significant differences between the groups (Fig. 2). Similarly, there were no statistical differences between the groups with regard to total body heat and change in total body heat in this period (Fig. 2). The mean skin temperatures in the radiant group were significantly higher than those in the other two groups throughout the postwarming period;  $p < 0.01$  throughout (Fig. 2).

**Shivering.** The radiant group showed less severe shivering than the other two groups; however, there were no significant differences in the shivering scores for the three groups

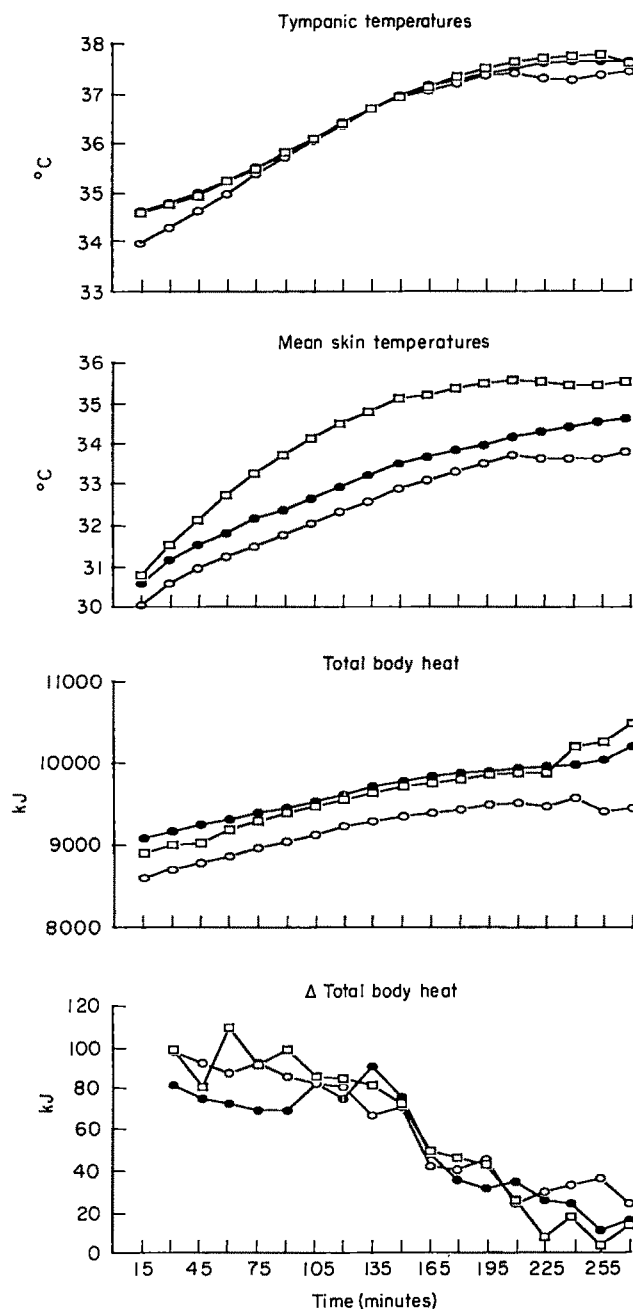


Fig. 1. Tympanic membrane temperature ( $^{\circ}\text{C}$ ), mean skin temperature ( $^{\circ}\text{C}$ ), total body heat (kJ) and  $\Delta$  total body heat (change of total body heat in the preceding 15 minutes) (kJ) for the whole of the study period. All data are presented as mean values. Control group, closed circles; oesophageal group, open circles; radiant group, open squares. Time 0 is the time of arrival the patient on the intensive care unit. The first recordings were 15 minutes later.

at any time interval (Fig. 3) or in the total shivering scores for the whole study period (Table 2).

### Discussion

Many studies have demonstrated that hypothermia is very difficult to avoid after cardiopulmonary bypass procedures.<sup>10-14</sup> Techniques aimed at preventing postoperative hypothermia include prolonged rewarming,<sup>6</sup> high bypass flows and vasodilator therapy during rewarming,<sup>15</sup> regulation of bypass flows to ensure muscle oxygenation<sup>13</sup> and

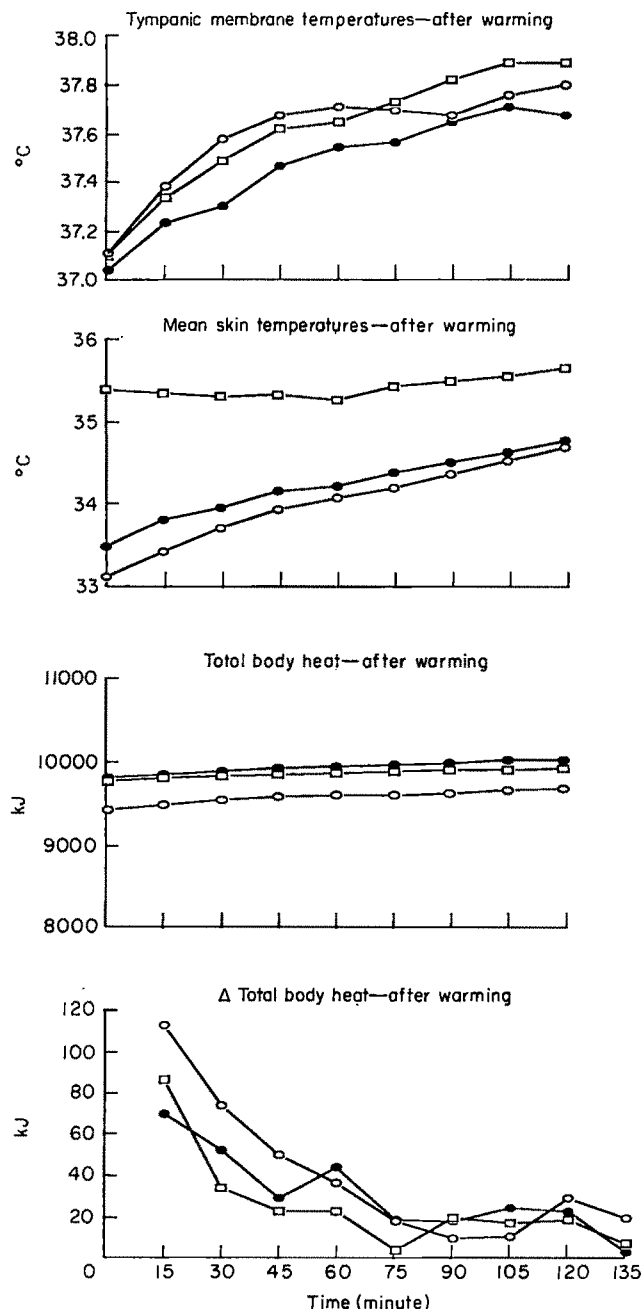


Fig. 2. Tympanic membrane temperature ( $^{\circ}\text{C}$ ), mean skin temperature ( $^{\circ}\text{C}$ ), total body heat (kJ) and  $\Delta$  total body heat (change of total body heat in the preceding 15 minutes) (kJ) during the post warming period. The post warming period began with the first tympanic membrane temperature greater than  $37^{\circ}\text{C}$  and continued for 2 hours thereafter. All data are presented as mean values. Control group, closed circles; oesophageal group, open circles; radiant group, open squares.

heated humidification of the inspired gases.<sup>16,17</sup> None of these techniques is totally successful, but if untreated, hypothermia in the postoperative period results in thermoregulatory efforts on the part of the patient that can produce potentially harmful increases in metabolic rate with increased oxygen consumption, carbon dioxide production and peripheral vasoconstriction.<sup>5,18</sup> Heavy sedation with opioids,<sup>19</sup> neuromuscular blockade<sup>20</sup> or active rewarming with radiant heaters<sup>4,5,21</sup> have previously been demonstrated to be effective methods of preventing shivering and thus the resulting cardiovascular and meta-



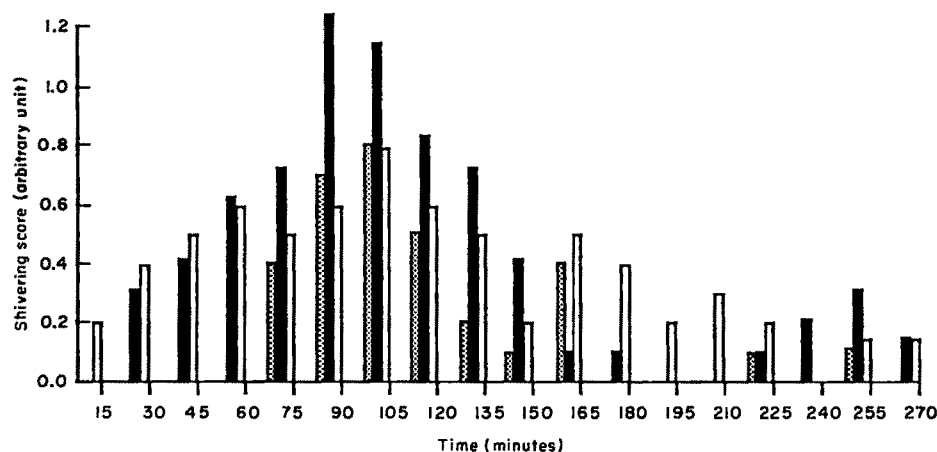


Fig. 3. Mean shivering scores during the whole of the study period (see methods for details). Control group, solid bars; oesophageal group, open bars; radiant group, hatched bars.

bolic changes. Sedation and myoneural blockade will both obtund the shivering response to hypothermia, slowing the rate of rewarming but allowing rewarming to occur at a normal metabolic rate. This will inevitably mean a longer period of postoperative ventilation.

External heat supply in the form of radiant overhead heaters will suppress thermoregulatory shivering by stimulation of the cutaneous thermoreceptors; therefore, intrinsic heat production by the patient is reduced while heat gain from the environment is increased. The rate of rewarming will depend on the balance of these two effects. The vasodilatation produced by cutaneous heating may also be of advantage in the postoperative patient after cardiopulmonary bypass. Radiant heaters, however, are not widely used in the UK, probably because they create an uncomfortable working environment for the nursing and medical staff and cannot easily be used in theatre during surgery.

Oesophageal rewarmers were first developed for use in the treatment of accidental hypothermia,<sup>2,22</sup> where they are claimed to be effective, although no controlled trials have been undertaken. *In vitro* tests show the calculated rate of heat transfer over the temperature range 19–37°C to be 339 kJ/hour.<sup>1</sup> This applied to a 70-kg man would result in a rate of increase of mean body temperature of 1.39°C/hour (assuming no losses to the environment). Thus, oesophageal rewarming devices promised to provide rapid postoperative warming without the environmental drawbacks of the radiant heaters. An additional benefit would be the use of the rewarming device in theatre after cessation of bypass to help minimise the afterdrop in body heat. The effect of these devices on shivering has never been studied, but potential problems resulting from their use include thermal injury to the oesophagus, local trauma to the oropharynx and oesophagus and leakage of fluid into the oesophagus (the Exacon device stops circulating water if more than 500 ml is lost from the system). The Exacon warmer proved to be simple to use and no complications associated with its use occurred during the course of the study.

This study set out to investigate the efficacy of the two methods of rewarming and to compare them with normal practice in this hospital, which is for no active rewarming to be employed. The only statistically significant findings in the study were the higher mean skin temperatures produced by radiant heat. It must be noted that the formula used, in

this and several other studies<sup>4,5,6,11</sup> to derive mean skin temperatures has not been validated during application of external heat sources and may overestimate values under these conditions. Shivering was reduced in the radiant group, tympanic membrane temperature increased slightly more rapidly in the oesophageal group and both treatment groups gained total body heat more rapidly than the control group. However, all differences were small and failed to reach statistical significance, but there are several possible reasons why the rewarming devices did not produce a clearer benefit. The ambient temperature in our ICU was relatively high and the patients were not profoundly hypothermic before the start of the trial. Both these factors will tend to minimise the temperature gradients over which the rewarming devices work and thus the heat transfer to the patients. The degree of shivering recorded in our study was low, indeed the control group in this study showed roughly the same degree of shivering as the treatment group in a recent study of radiant heating after cardiopulmonary bypass.<sup>5</sup> The reason for this is not clear, but may be because of different usage of sedative and vasodilator agents peri-operatively and the high ambient temperature in our ICU. Also, for reasons of modesty on our open plan ICU, the patients in this study were all nursed covered to the waist. This reduces the effective skin area available for radiant heating.

We have been unable to find any clear improvement in the rate of rewarming from the use of either method of heating. A large study may demonstrate small differences between the groups, but it is unlikely that this will be clinically significant. The mobile radiant heater was cumbersome to use and disliked by the medical and nursing staff, but may be useful if the ambient temperature in the ICU is low or shivering is a clinical problem. The Exacon thermal tube is expensive, invasive and does not suppress shivering and therefore cannot be recommended for routine use after cardiopulmonary bypass procedures.

## References

1. KRISTENSEN G, GRAVENSEN H, BENVENISTE D, JORDENING H. An oesophageal thermal tube for rewarming in hypothermia. *Acta Anaesthesiologica Scandinavica* 1985; 29: 846–8.
2. KRISTENSEN G, DRENK NE, JORDENING H. Simple system for central rewarming of hypothermic patients. *Lancet* 1986; 2: 1476.

3. KRISTENSEN G, GULDAGER H, GRAVENSEN H. Prevention of perioperative hypothermia in abdominal surgery. *Acta Anaesthesiologica Scandinavica* 1986; **30**: 314-6.
4. JOACHIMSSON P-O, NYSTROM S-O, TYDEN H. Heating efficacy of external heat supply during and after open-heart surgery with hypothermia. *Acta Anaesthesiologica Scandinavica* 1987; **31**: 73-80.
5. JOACHIMSSON P-O, NYSTROM S-O, TYDEN H. Postoperative ventilatory and circulatory effects of heating after aortocoronary bypass surgery. Postoperative external heat supply. *Acta Anaesthesiologica Scandinavica* 1987; **31**: 532-42.
6. JOACHIMSSON P-O, NYSTROM S-O, TYDEN H. Postoperative ventilatory and circulatory effects of extended rewarming during cardiopulmonary bypass. *Canadian Journal of Anaesthesia* 1989; **36**: 9-19.
7. RAMANATHAN NL. A new weighing system for mean surface temperature of the human body. *Journal of Applied Physiology* 1964; **19**: 531-3.
8. BURTON AC. The average temperature of the tissues of the body. *Journal of Nutrition*. 1935; **9**: 264-7.
9. COLIN J, TIMBAL J, HOUDAS Y, BOUTELIER C, GUIEU JD. Computation of mean body temperature from rectal and skin temperatures. *Journal of Applied Physiology* 1971; **31**: 484-9.
10. O'LEARY G, TEASDALE S. Prevention of shivering after cardiac surgery using radiant heat. *Canadian Journal of Anaesthesia* 1989; **36**: S90.
11. SHANKS CA, WADE LD, MEYER R, WILKINSON CJ. Changes in body temperature and heat in cardiac surgical patients. *Anaesthesia and Intensive Care* 1984; **13**: 12-7.
12. DONATI F, MAILLE J-G, BLAIN R, BOULANGER M, SAHAB P. End-tidal carbon dioxide tension and temperature changes after coronary artery bypass surgery. *Canadian Anaesthetists' Society Journal* 1985; **32**: 272-7.
13. STANLEY TH, JACKSON J. The influence of blood flow and arterial blood pressure during cardiopulmonary bypass on deltoid muscle gas tensions and body temperature after bypass. *Canadian Anaesthetists' Society Journal* 1979; **26**: 277-81.
14. SLADEN RN. Temperature and ventilation after hypothermic cardiopulmonary bypass. *Anesthesia and Analgesia* 1985; **64**: 816-20.
15. NOBACK CR, TINKER J. Hypothermia after cardiopulmonary bypass in man. Amelioration by nitroprusside-induced vasodilatation during rewarming. *Anesthesiology* 1980; **53**: 277-80.
16. CALDWELL C, CRAWFORD R, SINCLAIR I. Hypothermia after cardiopulmonary bypass in man. *Anesthesiology* 1981; **55**: 86-7.
17. RALLEY FE, RAMSAY JG, WYNANDS JE, TOWNSEND GE, WHALLEY DG, DELICOLLI P. Effect of heated humidified gases on temperature drop after cardiopulmonary bypass. *Anesthesia and Analgesia* 1984; **63**: 1106-10.
18. RALLEY FE, WYNANDS JE, RAMSAY JG, CARLI F, MACSULLIVAN R. The effects of shivering on oxygen consumption and carbon dioxide production in patients rewarming from cardiopulmonary bypass. *Canadian Journal of Anaesthesia* 1988; **35**: 332-7.
19. RODRIGUEZ JL, WEISSMAN C, DAMASK MC, ASKANAZI J, ALLEN IH, KINNEY JM. Morphine and postoperative rewarming in critically ill patients. *Circulation* 1983; **68**: 1238-46.
20. RODRIGUEZ JL, WEISSMAN C, DAMASK MC, ASKANAZI J, HYMAN AI, KINNEY JM. Physiologic requirements during rewarming; suppression of the shivering response. *Critical Care Medicine* 1983; **11**: 490-7.
21. SHARKEY A, LIPTON JM, MURPHY MT, GIESECKE AH. Inhibition of postanesthetic shivering with radiant heat. *Anesthesiology* 1987; **66**: 249-52.
22. LEDINGHAM IM, DOUGLAS IHS, ROUTH GS, MACDONALD AM. Central rewarming system for treatment of hypothermia. *Lancet* 1980; **1**: 1168-9.

## Dr F. P. de Caux—the first user of curare for anaesthesia in England

D. J. WILKINSON

### Summary

*Curare was used in the 19th century in England by a wide variety of scientists, physicians and veterinarians. Their experiments indicated many of the properties of the drug, but its clinical usage remained very limited and was reserved for cases of tetanus, hydrophobia and strychnine poisoning. Griffith and Johnson are usually credited with the introduction of curare into clinical anaesthesia in 1942, but a Dr F.P. de Caux working at the North Middlesex Hospital, London, in 1928 utilised curare in a series of seven patients. His work was not widely publicised and this contribution to anaesthetic history has been overlooked by subsequent authors.*

### Key words

History; F. P. de Caux.  
Neuromuscular relaxants; curare.

The use of curare by South American Indians has been reported in Europe since the early 16th century. Pieter Angherius's book, *De orbe novo*, published in 1505 initially and then translated into English by Richard Eden in 1555 is probably the earliest text that refers to this poison.<sup>1</sup> Various explorers to South America returned with quantities of curare some of which was subjected to investigation.<sup>2–4</sup> Animal experiments by Brodie,<sup>5</sup> Waterton<sup>6</sup> and Sewell<sup>7</sup> in the early 19th century renewed interest in curare which was then advocated for use in the treatment of strychnine poisoning, hydrophobia and tetanus. Sporadic reports on its use in this field appear in the world literature over the next hundred years.<sup>8–11</sup>

The first use of muscle relaxants in anaesthesia took place in 1912 in Leipzig when Lawen advocated its use during a balanced anaesthetic technique to facilitate abdominal wound closure. His supply of the drug was limited and his report did not attract widespread interest.<sup>12</sup>

### Dr F. P. De Caux

Francis Percival de Caux was born in Takaka, New Zealand on 1892 and moved to England in 1912. He qualified in medicine from St Bartholomew's Hospital in

1921 and started to specialise in anaesthesia almost immediately. He held several honorary anaesthetic posts including ones at the Royal Dental Hospital and Kensington Hospital before joining the staff of the North Middlesex Hospital in 1927.

His major interest was in nitrous oxide and oxygen anaesthesia particularly for dental surgery, but he perfected its use for major surgery as well. He designed several new pieces of apparatus<sup>13,14</sup> and produced a modification of the Mckesson demand flow nitrous oxide/oxygen apparatus manufactured by A. Charles King. He was described by Sir Ivan Magill as 'the finest exponent of nitrous oxide/oxygen anaesthesia that I ever saw.' de Caux published extensively on his work with nitrous oxide in dental anaesthesia<sup>15,16</sup> but was fully aware of its limitations in producing deep anaesthesia.

### Curare

The only reference to de Caux's researches with curare appear in Dr Victor Goldman's textbook *Aids to anaesthesia*. There is no reference to muscle relaxants in the first edition of 1941, but the second edition of 1948 has a full chapter on curare, which begins with an historical review.<sup>17</sup>

---

Dr D. J. Wilkinson, MB, BS, FFARCS, Consultant Anaesthetist, Department of Anaesthesia, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE.

Part of this work was presented at the IXth World Congress of Anaesthesiology, Washington 1988 and also at the History of Anaesthesia Society Meetings in Southend and Leicester in the same year.

Accepted 30 April 1990.



I have known F. P. de Caux since 1926, and from then until 1939 he was anaesthetist at the North Middlesex County Hospital where I was surgeon and he gave many thousands of superlative anaesthetics for me in that service; I may incidentally state that he was the first person in this country to use Curare as an adjuvant in Gas and Oxygen anaesthesia, and this was in the year 1928 when it was administered to seven cases, but, owing to difficulties in procuring physiological standardised Curare, the experiment lapsed.

Fig. 3. Reference for Dr de Caux from a North Middlesex Hospital surgeon.

matters—I suppose owing to Quaker upbringing, and would not like me for writing such a letter about himself.

To both my best wishes.

Yours very sincerely

Frank Coleman'

A fascinating letter; his father Alfred Coleman, also from St Bartholomew's Hospital, has certainly received full acclaim since his work on CO<sub>2</sub> absorption.<sup>18</sup> de Caux's response to this letter is typical of his honesty and modesty (Fig. 2) and can be clearly read. Ranyard West used a similar product in 1932.<sup>19</sup> He states he obtained curare from a Sir Charles Sherrington 'It was a resinous mass of the consistency of hard toffee, and was incompletely soluble in water, Messrs Burroughs Wellcome filtered it and sterilised it by autoclaving and supplied me with the drug in ampoules of suitable strength.' Further evidence in support of de Caux comes from another reference written in the early 1950s (Fig. 3) which repeats the story.

F. P. de Caux was in the forefront of anaesthetic research at this time, particularly in his favoured field of nitrous oxide anaesthesia. He was well known internationally and frequently travelled in Europe where he met and exchanged ideas with other anaesthetists, particularly in Czechoslovakia. It may be that it was one such visit that brought the early German experience with curare to his attention and thus instigated his own researches.

### Conclusion

There is now further documentary proof of the use of curare during anaesthesia by Dr F. P. de Caux in 1928 at the North Middlesex Hospital. This supports the only previously documented claim by Dr Victor Goldman which is in turn supported by his own personal recollection of that time. Dr de Caux's failure to publish his results has meant that his contribution in this area of anaesthetic research has been overlooked. His work with curare did little to influence its later acceptance by his profession, although it nevertheless represents the first use of the drug in the UK during anaesthesia. de Caux's contribution to our specialty deserves wider recognition.

### Acknowledgment

I thank Mrs de Caux, Professor P. de Caux, and Dr Victor Goldman for their considerable assistance in my

researches. I am grateful to the Department of Medical Illustration at St Bartholomew's Hospital for the photographs provided and to Miss M. Langley for considerable secretarial assistance.

### References

1. ANGERIUS PM. *De orbe novo*. Translated by Richard Eden, London, 1555.
2. Condamine, de la. Relation abregée d'un voyage dans l'intérieur de l'Amerique Meridionale. *Transactions de l'academie des Sciences* 1745; **62**: 391
3. BANCROFT E. An essay on the natural history of Guiana and South America. London: T. Becket and P. A. De Hont, 1769.
4. FONTANA F. On the American poison called Ticunas. *Philosophical Transactions*, 1780; **70**: 163.
5. BRODIE BE. Experiments and observations on the different modes in which death is produced by certain vegetable poisons. *Philosophical Transactions* 1811; **101**: 178.
6. WATERTON CH. Wanderings in South America. London: Mawman, 1835.
7. TRAVERS B. A further inquiry concerning constitutional irritation. London: Longman Hurst Rees, 1835.
8. VULPIAN A. De l'emploi de curare comme antidote de la strychnine et comme traitement tetanos. *L'Union med. Paris*, 1857; **1**: 25-6.
9. SAYRES LA. Two cases of traumatic tetanus. *New York Journal of Medicine* 1858; **4** 250-3.
10. HARLEY G. On the action of woorara. *Lancet* 1859; **2**: 654.
11. M'ARDLE JS. The treatment of acute traumatic tetanus by curare. *Dublin Journal of Medical Science* 1886; **81**: 123-8.
12. LAWEN A. Ueber die Verbindung der Localanaesthesie mit der Narkose, über hohe Extraduralanästhesie und epidurale Injectionen an ästhesierender Lösungen bei tabischen Magenkrise, *Beiträge zur klinischen chirurgie*, 1912; **80**: 168-89.
13. DE CAUX FP. A new pattern oxygen sight feed valve. *Lancet* 1923; **2**: 228.
14. DE CAUX FP. Closed drop ether inhaler. *Lancet* 1923; **2**: 789-90.
15. DE CAUX FP. Endotracheal gas and oxygen for dental operations. *The Ivory Cross Journal* 1924; **10**: 643-5.
16. DE CAUX FP. Recent advances in methods of administering nitrous oxide in oral surgery. *British Dental Journal* 1930; **51**: 471-84.
17. GOLDMAN V. *Aids to anaesthesia*, 2nd edition. London: Baillière Tindall and Cox, 1848: 118-9.
18. FOREGGER R. Early use of the carbon dioxide absorption method in anaesthetic practice. *British Journal of Anaesthesia* 1961; **33**: 470-5.
19. WEST R. Curare in man. *Proceedings of the Royal Society of Medicine* 1932; **25**: 39-48.

## Forum

### Minimum oxygen requirements during anaesthesia with the Triservice anaesthetic apparatus

#### A study of drawover anaesthesia in the young adult

S.Q.M. Tighe,\* MB, BS, FFARCS, RN, Senior Registrar, G.A. Turner, MB, BS, FFARCS, RN, Consultant, Department of Anaesthetics, The Royal Naval Hospital Haslar, Gosport, Hampshire, PO12 2DH, S.B. Merrill, MB, BS, FFARCS, RN, Consultant, Department of Anaesthetics, The Royal Naval Hospital Plymouth, Plymouth, Devon, PL1 3JY, R.J. Pethybridge, BSc, PhD, Principal Statistician, The Institute of Naval Medicine, Alverstoke, Gosport, Hampshire PO12 2DP.

#### Summary

Thirty-six servicemen were anaesthetised using the Triservice anaesthetic apparatus. They were allocated randomly into one of two groups, to breathe spontaneously or to receive artificial ventilation, and into subgroups who were given air alone, or air supplemented with 1 or 4 litres/minute of oxygen. A further 12 subjects were studied subsequently using 0.5 litres/minute of added oxygen. Intra-operative blood gases were compared with those of awake premedicated controls. Artificial ventilation was associated with an unchanged arterial oxygen tension with air alone; in the other subgroups arterial oxygen tension was higher than with spontaneous respiration when related to inspired oxygen fraction ( $p < 0.05$ ). Air anaesthesia caused significant hypoxaemia with spontaneous ventilation ( $p < 0.05$ ), and 50% of the subjects required assisted ventilation. There was also a significant respiratory acidosis ( $p < 0.05$ ). Intermittent positive pressure ventilation is the method of choice for field anaesthesia when oxygen is unavailable. Spontaneous respiration must be supplemented with at least 0.5 litres/minute of oxygen.

#### Key words

Oxygen; blood levels.

Equipment; Triservice anaesthetic apparatus.

Compressed oxygen ( $O_2$ ) was a scarce commodity during the Falklands war, and air alone was used commonly during drawover anaesthesia<sup>1</sup> with the Triservice anaesthetic apparatus (TSA).<sup>2</sup> This might be considered a dangerous practice since it is widely accepted that oxygen desaturation occurs during anaesthesia if air is administered.

The literature is contradictory as to the relative merits of spontaneous ventilation (SV) and intermittent positive pressure ventilation (IPPV) in terms of oxygenation during conventional anaesthesia<sup>3–9</sup> and there have been no comparative reports of halothane/air anaesthesia in a drawover system in this respect.

This study aimed to establish the minimum flow of oxygen supplementation associated with acceptable arterial oxygen tensions in a comparison of IPPV with SV using the TSA technique.<sup>10</sup> In this way, recommendations could be made that would not only ensure the safe and economical use of oxygen in the field, but would also have some relevance in the civilian context.

#### Method

Thirty-six ASA 1 servicemen were studied initially. They were aged 18–41 years, weighed less than 100 kg and were scheduled for minor orthopaedic surgery. The protocol was

approved by the local ethics committee and informed consent was obtained.

Subjects were allocated randomly into six subgroups, in which the patients received 0, 1 or 4 litres/minute of intra-operative oxygen supplementation and either SV or IPPV.

Premedication was with intramuscular papaveretum 0.3 mg/kg, prescribed one hour pre-operatively. A premedication–induction interval of 30–90 minutes was allowed.

Four arterial pressure recordings were made at 2.5 minute intervals before induction using a Datascope Accutor 1 automatic analyser. Ear lobe oxygen saturation ( $SpO_2$ ) was recorded using an Ohmeda Biox 1 oximeter. An arterial blood sample was taken from the femoral artery and analysed immediately in an Automated Biochemistry Laboratories series 4 blood gas analyser; results were corrected for body temperature.

Anaesthesia was induced with thiopentone 5 mg/kg. Subjects randomised to the SV group received suxamethonium 1 mg/kg and those in the IPPV group, vecuronium 0.1 mg/kg. Manual ventilation at a rate of 12 breaths/minute with air supplemented by 4 litres/minute of oxygen was initiated and intubation performed at 30 or 120 seconds respectively. Indicated concentrations of halothane and trichloroethylene were set at 3% and 1% for the first 3 minutes, and reduced to 1.5% and 0.5% respectively. Manual ventilation was continued in the SV groups until

Accepted 3 May 1990.

\*Now Consultant Anaesthetist at the same address.



spontaneous ventilation resumed, whereas in the IPPV groups ventilation was adjusted to maintain an end-tidal partial pressure of carbon dioxide ( $P_{E'}CO_2$ ) of 4.6 kPa using a Hewlett Packard 78345A capnograph. A tidal volume of 10 ml/kg delivered by a Cape TC 50 ventilator was set and the rate adjusted accordingly.

The oxygen flow was then adjusted, to 0, 1 or 4 litres/minute after calibrating the  $O_2$  Rotameter of a Boyle's machine with a Timeter Instrument Corporation RT 200 calibration analyser.

Indicated concentrations of volatile agents were subsequently adjusted according to clinical requirements.

The electrocardiogram,  $SpO_2$ ,  $P_{E'}CO_2$ , and fractional inspired oxygen concentration ( $F_{IO_2}$ ) were monitored continuously, the last with a Hewlett Packard 78345A polarographic oxygen meter. The heart rate and arterial blood pressure were recorded at 2.5-minute intervals with the Datascope analyser, together with the above indices, the respiratory rate and the tidal volume (Wright's respirometer).

A second arterial blood sample was taken from the nondominant radial artery at least 10 minutes after the surgical incision and after at least 10 minutes of stable  $SpO_2$  ( $\pm 1\%$ ) and minute volume recordings. The sample was analysed immediately for blood gas concentrations, which were corrected for body temperature. The  $F_{IO_2}$  (end-inspiration) and  $P_{E'}CO_2$  at the time of sampling were also recorded.

IPPV was instituted in the SV group in the following circumstances: if  $SpO_2$  decreased to less than the pre-operative control value and was associated with either hypotension ( $< 75\%$  of the pre-operative mean), an arrhythmia or hypercapnia ( $> 8.0$  kPa); any other combination of the above; at the discretion of the anaesthetist.

Oxygen was administered at a rate of 4 litres/minute if there was no improvement, or if the above circumstances occurred in the IPPV group. A further arterial sample was taken when the initiating event had resolved.

Administration of trichloroethylene was discontinued at an estimated 15 minutes before the completion of surgery and oxygen was administered at 4 litres/minute before extubation. Neostigmine 2.5 mg and atropine 1.2 mg were administered to patients in the IPPV groups.

The following time intervals were measured: premedication to induction; premedication to first blood gas analysis; premedication to second blood gas analysis; first to second blood gas analysis; induction to incision; incision to completion of surgery; induction to extubation; and extubation to correct recall of name, rank and number.

The alveolar oxygen partial pressure ( $PAO_2$ ) was calculated from the simplified alveolar air equation;<sup>13</sup>  $PAO_2 = P_{IO_2} - PaCO_2/R$ , where  $P_{IO_2}$  is the partial pressure of oxygen at sea level and R is the respiratory quotient (0.8). The alveolar-arterial oxygen partial pressure difference was then calculated ( $P(A-a)O_2$ ).

At the conclusion of this trial, a further 12 patients were studied using air supplemented by 0.5 litres/minute of oxygen, and were randomised to receive either SV or IPPV. The method was otherwise as described above.

Equipment was calibrated appropriately before each patient was studied and the same items were used throughout. All equipment was also calibrated and serviced by the manufacturers before the trial was started. Figure 1 illustrates the apparatus layout.

Statistical analysis was by analysis of variance (ANOVA) incorporating the two main factors (oxygen and ventilation), the third factor (repeated measures on the same subjects-time) and the interactions (three pairs and one triple). Subjects were assumed to be a random sample from all possible patients who would fulfil the inclusion criteria. The Kruskal-Wallis nonparametric test was employed to assess if any differences occurred with demographic indices or time intervals.

## Results

Observations were obtained on 18 patients in each group (SV and IPPV), and on six patients per subgroup (0, 1 and 4 litres/minute of oxygen). An additional 12 patients were studied at an oxygen flow rate of 0.5 litres/minute (6 SV and 6 IPPV).

There were no significant differences among subgroups in respect of age, weight or smoking habits (Table 1). The mean age was 28 (range 18-41) years.

There were no significant differences among subgroups in any time intervals.

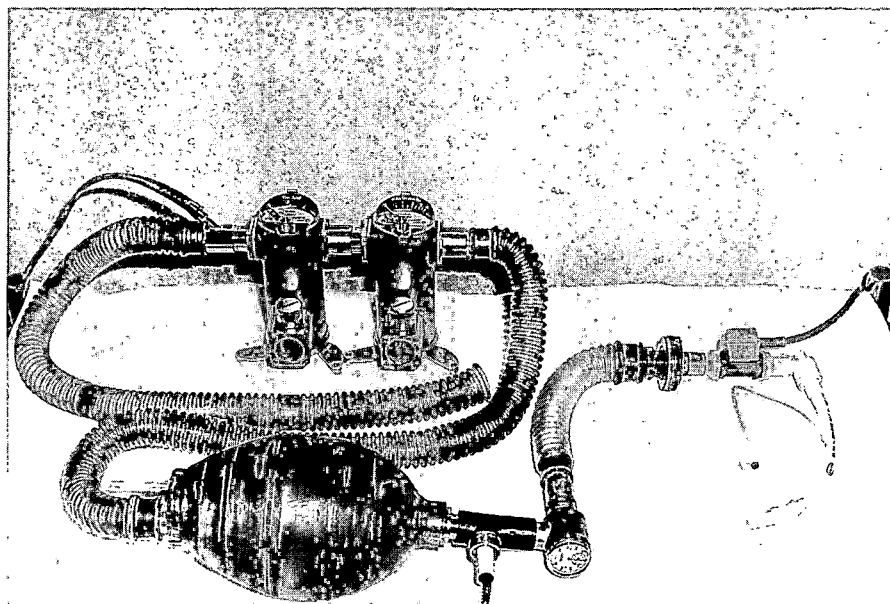


Fig. 1. The Triservice Anaesthetic Apparatus, as used in this study during spontaneous ventilation. The Cape TC50 ventilator replaced the Laederal bag during IPPV.

Table 1. Mean (range) demographic indices.

O <sub>2</sub> subgroup (litres/minute)	Ventilation group							
	SV				IPPV			
	0	1	4	All	0	1	4	All
Age; years	25 (18–36)	26 (22–38)	33 (23–41)	28 (18–41)	27 (20–40)	30 (25–35)	27 (18–41)	28 (18–41)
Weight; kg	80 (67–97)	80 (71–93)	76 (70–84)	79 (67–97)	80 (65–92)	77 (58–95)	78 (57–100)	78 (57–100)
Smokers (n)	1	3	4	8	2	1	3	6

SV, spontaneous ventilation; IPPV, intermittent positive pressure ventilation.  
n=6 for each subgroup.

The mean respiratory rates were 25.2 (range 13.9–37.3) and 11.0 (range 8.4–14.7) breaths/minute for SV and IPPV groups respectively; the mean difference of 14.2 was significant ( $p < 0.01$ , 95% confidence interval for differences of means (CI) 11.2–17.2). The corresponding figures for minute volume were 5.1 (range 2.6–9.2) and 6.7 (range 5.0–10.2) litres/minute, a significant average difference of 1.6 ( $p < 0.01$ , 95% CI 0.6–2.8).

There was a significant ( $p < 0.05$ ) respiratory acidosis in the SV group when compared to both controls and the IPPV group at all oxygen flow rates (Table 2). The mean changes of pH and  $P_{aCO_2}$  from control were  $-0.074$  pH units (95% CI  $-0.094$  to  $-0.043$ ) and  $1.33$  kPa (95% CI  $0.92$ – $1.73$ ) respectively in the SV group. The corresponding figures were  $0.047$  pH units (95% CI  $0.027$ – $0.067$ ) and  $-0.72$  kPa (95% CI  $-1.01$  to  $-0.43$ ) in patients who received IPPV, reflecting a significant, mild respiratory alkalosis ( $p < 0.05$ ).

IPPV was associated with a lower mean  $F_{IO_2}$  than SV at both O<sub>2</sub> flow rates (Table 3) but this was significant only in the 1 litre/minute oxygen subgroup ( $p < 0.05$ ), with a mean difference of  $0.07$  (95% CI  $0.01$ – $0.13$ ). At 4 litres/minute, the mean difference was  $0.05$  (95% CI  $-0.12$ – $0.22$ ).

Air anaesthesia with SV caused significant hypoxaemia (mean  $P_{aO_2}$  8.8, range 6.6 to 12.8 kPa), when compared to both controls (mean  $P_{aO_2}$  12.2, range 10.7 to 15.0 kPa, an average difference of  $3.4$  kPa, 95% CI  $1.3$ – $5.6$ ,  $p < 0.05$ ), and to the IPPV group (mean  $P_{aO_2}$  12.3, range 10.0 to 15.0 kPa, an average difference of  $3.5$  kPa, 95% CI  $1.2$ – $5.8$ ,  $p < 0.05$ ) (Table 3, Fig. 2). Three subjects (one smoker)

required the institution of IPPV which restored the  $P_{aO_2}$  to a mean of  $11.4$  kPa; this was not significantly different from his baseline value. The use of IPPV with unsupplemented air was associated with a  $P_{aO_2}$  that was insignificantly different from baseline.

There were no significant differences in  $P_{aO_2}$  between groups and subgroups when oxygen was added. However, when  $P_{aO_2}$  is adjusted for  $F_{IO_2}$  (Fig. 2), a difference between SV and IPPV groups is apparent. When applied to all oxygen subgroups, analysis yields parallel  $P_{aO_2}$   $F_{IO_2}$  relationships, with a common slope of 60 and a vertical separation of  $3.8$  kPa (95% CI  $0.1$ – $7.4$ ,  $p < 0.05$ ), with IPPV higher than SV. A smoking effect was suspected, but this could not be defined clearly. Nevertheless, when smokers were eliminated from the analysis, a significant difference between IPPV and SV groups was still apparent, with a vertical separation of  $4.7$  kPa (95% CI  $1.1$ – $8.3$ ,  $p < 0.05$ ). There were no significant differences in  $P_{aO_2}$  between smokers and nonsmokers.

There was a consistent significant difference in mean  $P_{aO_2}$  between IPPV and SV groups when related to  $F_{IO_2}$  as above (Table 3, Fig. 2), with a common slope of 95 and a vertical separation of  $2.5$  kPa (95% CI  $1.9$ – $3.0$ ,  $p < 0.01$ );  $P_{aO_2}$  was higher during IPPV than SV. After adjusting for

Table 2. Mean (SEM) change of intra-operative pH,  $P_{aCO_2}$  and base excess (BE) from control.

Ventilation group	O <sub>2</sub> subgroup (litres/minute)	pH	$P_{aCO_2}$ (kPa)	BE (mmol/litre)
SV	0	$-0.063^*$ (0.016)	$1.25^*$ (0.35)	$-0.87^*$ (0.29)
	1	$-0.062^*$ (0.021)	$1.19^*$ (0.41)	$-0.18$ (0.55)
	4	$-0.097^*$ (0.012)	$1.54^*$ (0.27)	$-1.65^*$ (0.33)
	0	$0.046$ (0.017)	$-0.67^\dagger$ (0.25)	$0.18$ (0.53)
IPPV	1	$0.074$ (0.009)	$-0.98^\dagger$ (0.14)	$0.12$ (0.32)
	4	$0.021$ (0.018)	$-0.52$ (0.30)	$-0.53$ (0.41)

SV, spontaneous ventilation; IPPV, intermittent positive pressure ventilation.

n=6 for each subgroup.

\* $p < 0.05$ , compared with both baseline and IPPV (ANOVA).

† $p < 0.05$ , compared with baseline (ANOVA).

Table 3. Mean (SEM)  $P_{aO_2}$ ,  $P_{aO_2}$ ,  $P(A-a)O_2$ , and  $F_{IO_2}$ .

O <sub>2</sub> subgroup (litres/minute)	Ventilation group					
	SV			IPPV		
	0	1	4	0	1	4
Intra-operative	(0.6)	(0.7)	(0.3)	(0.3)	(0.6)	(0.8)
$P_{aO_2}$ (kPa)	8.8* (0.7)	18.9 (2.7)	39.0 (5.4)	12.3 (0.7)	18.7 (1.5)	43.1 (6.8)
Baseline	13.2 (0.3)	13.3 (0.4)	13.1 (0.2)	13.0 (0.2)	13.1 (0.2)	13.2 (0.2)
Intra-operative	11.6 (0.5)	29.6 (2.4)	63.4 (6.6)	13.9 (0.2)	25.5 (0.5)	61.5 (3.5)
$P(A-a)O_2$ (kPa)	1.0 (0.5)	1.2 (0.5)	1.8 (0.2)	1.5 (0.3)	1.2 (0.5)	1.2 (0.7)
Baseline	2.8 (0.4)	10.6 (1.7)	24.4 (4.9)	1.6 (0.6)	6.8 (1.8)	18.4 (4.9)
$F_{IO_2}$	0.21 (0.00)	0.40 (0.03)	0.76 (0.07)	0.21 (0.00)	0.33† (0.01)	0.72 (0.04)

SV, spontaneous ventilation; IPPV, intermittent positive pressure ventilation.

n=6 for each subgroup.

\* $p < 0.05$ , compared to both baseline and IPPV (log conversion, ANOVA).

† $p < 0.05$ , compared with SV (ANOVA).

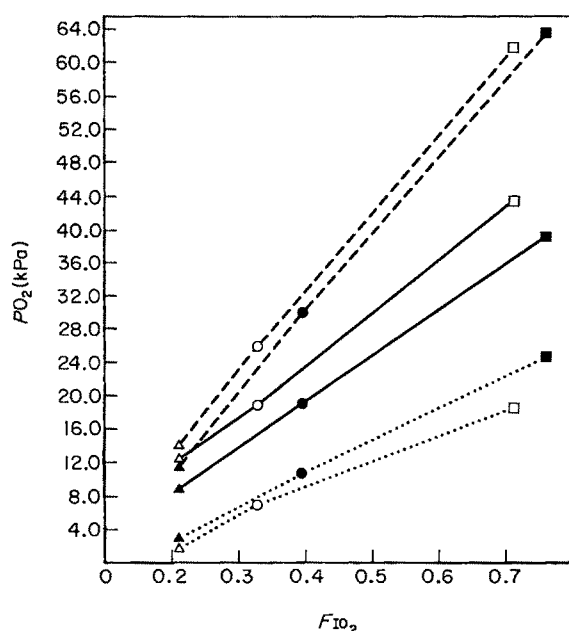


Fig. 2. Mean  $PAO_2$  (---),  $Pao_2$  (—) and  $P(A-a)O_2$  (....) plotted against mean  $FIO_2$  at oxygen flow rates of 0, 1 and 4 litres/minute  $\blacktriangle, \bullet, \blacksquare$  for spontaneous ventilation and  $\triangle, \circ, \square$  for intermittent positive pressure ventilation respectively,  $n = 6$  for each subgroup.  $PAO_2$  and  $Pao_2$  are significantly higher with IPPV ( $p < 0.01$  and  $< 0.05$  respectively) after allowing for  $FIO_2$  in a regression model.

$FIO_2$ , the mean  $P(A-a)O_2$  was insignificantly greater in the SV group by 2.3 kPa (95% CI  $-2.4-7.0$ ).

Mean  $Pao_2$  with 0.5 litres/minute oxygen was 12.9 kPa (range 10.3–15.4) in the SV group and 13.31 kPa (range 10.8–15.7) during IPPV. This small difference between groups was not significant. With IPPV, mean  $Paco_2$  and pH were not significantly different from baseline. The minute volume was insignificantly different and the mean  $FIO_2$  was 0.27 in both groups. All other variables reflected the same differences between groups as described above.

### Discussion

This study was designed to simulate field anaesthesia as closely as is possible within the hospital environment. The surgery was relatively minor and peripheral since this represents the military experience.<sup>11</sup> Our results are not valid for more major procedures, when greater oxygen requirements can be expected.<sup>12</sup>

Shunt and ventilation-perfusion inequality contribute to an increased  $P(A-a)O_2$  during anaesthesia, regardless of the ventilation method.<sup>3,4</sup> Previous studies which compared oxygenation achieved with SV and IPPV have been inconclusive as to which technique results in less desaturation,<sup>5-9</sup> but in common with numerous reports which have examined one or other mode alone,<sup>13</sup> these comparisons are difficult to interpret because of the widely differing methodology.

Respiratory depression and acidosis were evident with SV at all oxygen flows (Table 2), and can be attributed to opioid premedication, the requirement for relatively high concentrations of volatile agent and the apparatus resistance. Houghton<sup>2</sup> states that the last is not relevant in clinical practice, but from his paper it is possible to estimate a pressure drop of 0.3–0.5 kPa at inspiratory flow rates typical of spontaneous ventilation (20–30 litres/minute). This is excessive and therefore clinically relevant, particularly when considering the additive effect of a

tracheal tube.<sup>14</sup> Hyperventilation with IPPV eliminated the possibility of hypercapnia; a  $Pe/CO_2$  of 4.6 kPa was selected because the TSA technique relies on manual ventilation at present and there is a natural tendency to hyperventilate.<sup>13</sup>

SV with unsupplemented air was associated with significant hypoxaemia (Table 3); 50% of subjects required IPPV, which improved the  $Pao_2$  to baseline values. This hypoxic effect of halothane/air anaesthesia has been noted previously with SV<sup>15</sup> but its prevention or reversal by IPPV has not.  $Pao_2$  was slightly above the baseline level during IPPV.

Hypocapnia increases the  $PAO_2$  according to the alveolar air equation.<sup>13</sup> However, because the minute volume of ventilation was significantly greater, the  $FIO_2$  at each oxygen flow rate was lower with IPPV than with SV, (Table 3, Fig. 2), according to the formula of Mackie,<sup>16</sup> tending to counteract the effect of hypocapnia on  $PAO_2$ . This explains why  $PAO_2$  and  $Pao_2$  were similar in SV and IPPV groups at each level of additional oxygen, but were significantly different when related to  $FIO_2$ . The use of IPPV at a given  $FIO_2$ , results in a higher  $Pao_2$  than SV. It is suggested that this is due both to the significant increase in  $PAO_2$  which results from hypocapnia and to the insignificantly lower  $P(A-a)O_2$  in almost equal proportion, but a larger study is necessary to prove this statistically. However, the result of these changes is clinically relevant only during unsupplemented air anaesthesia with the TSA, since oxygen flow is otherwise fixed, so that the reduction in  $FIO_2$  with IPPV and hyperventilation compensates for any tendency to increase  $Pao_2$ . In normal hospital practice, the differences between SV and IPPV might be more important since  $FIO_2$  is not affected by minute volume in conventional plenum anaesthetic systems, so that a lower  $FIO_2$  than is customarily administered might be acceptable with IPPV in a similar population.

We have assumed a value of 0.8 for R in the calculation of  $PAO_2$ , which may not be valid. However, any resulting error is likely to have been spread equally throughout the study. We could also be criticised for not having used a rapid-response oxygen analyser for measurement of  $FIO_2$ . The calculations of Mackie<sup>16</sup> show that the only variables to affect  $FIO_2$  with the TSA in clinical use are the oxygen flow and the minute volume. Other potential variables are irrelevant at the high respiratory rates seen with SV. This is true also for IPPV when a large oxygen reservoir is employed and when the ratio of inspiration to expiration is 1:1, as delivered by the Cape TC 50 ventilator. Both minute volume and oxygen flow rate had been stable for at least 10 minutes at the time  $FIO_2$  was recorded, and thus a rapid response analyser was unnecessary.

The relative youth of our subjects may be relevant since  $Pao_2$  in the awake subject decreases with age.<sup>17</sup> It would be logical to assume that this trend is maintained or even increased during anaesthesia, but surprisingly this has not been demonstrated conclusively. Nunn's classic study<sup>4</sup> found that all subjects less than 42 years of age who received IPPV and an  $FIO_2$  near 0.21 had an intra-operative  $Pao_2$  greater than the mean control and higher than 13 kPa. However, baseline blood gases were not analysed in all cases, the numbers in this age group were small and the calculated venous admixture did not increase significantly with age, although p was very close to 0.05. A minimum  $FIO_2$  of 0.31 was recommended for IPPV due to the wide variation of intra-operative  $Pao_2$ , but it would seem that this variation was most evident in the older population. A further study to compare the young with older age groups would be useful since previous publications have been inconclusive and have failed to show statistical significance.<sup>4,6,18,19</sup> Our results support the contention that the young and fit do not suffer from significant oxygen

desaturation during anaesthesia using air, moderate hyperventilation and IPPV.

Smoking might decrease  $P_{aO_2}$ ,<sup>20</sup> but the relationship between smoking habit and  $P_{aO_2}$  could not be clearly defined in this study because of the small number of smokers in various subgroups. There was some indication of different  $P_{aO_2}/F_{iO_2}$  relationships between smokers and nonsmokers, but a larger study is necessary to define these. Nevertheless, there was a significant difference in the  $P_{aO_2}/F_{iO_2}$  relationship between SV and IPPV groups when the data from smokers was eliminated from the analysis.

The TSA technique is intended for military use, and it is essential to consider the additional effects of hypovolaemia. Alveolar deadspace is increased, but venous shunting is unaffected during haemorrhage.<sup>21</sup> Hyperventilation with IPPV can compensate for increased deadspace and maintain  $PAO_2$ , whereas respiratory depression during SV results in further hypercapnia, acidosis and hypoxaemia in a situation where  $O_2$  delivery is already critical. This reason alone, quite apart from the question of oxygen economy, is sufficient to recommend the use of IPPV during anaesthesia for military casualties, provided that cardiac output is not compromised by excessive airway pressures. The more critical indices of tissue oxygen supply and consumption have not been assessed in this context, so that any theoretical advantage of IPPV must remain conjectural.

#### Acknowledgments

The authors thank their anaesthetic and orthopaedic colleagues for their patience and cooperation and Mr R. Howard for his technical assistance.

#### References

1. JOWITT MD. Anaesthesia ashore in the Falklands. *Annals of the Royal College of Surgeons of England* 1984; **66**: 197–200.
2. HOUGHTON IT. The Triservice anaesthetic apparatus. *Anaesthesia* 1981; **36**: 1094–1108.
3. NUNN JF. Factors influencing the arterial oxygen tension during halothane anaesthesia with spontaneous respiration. *British Journal of Anaesthesia* 1964; **36**: 327–41.
4. NUNN JF, BERGMAN NA, COLEMAN AJ. Factors influencing the arterial oxygen tension during anaesthesia with artificial ventilation. *British Journal of Anaesthesia* 1965; **37**: 898–914.
5. STARK DCC, SMITH H. Pulmonary vascular changes during anaesthesia. *British Journal of Anaesthesia* 1960; **32**: 460–5.
6. MARSHALL BE, MILLAR RA. Some factors influencing postoperative hypoxaemia. *Anaesthesia* 1965; **20**: 408–28.
7. PRICE HL, COOPERMAN LH, WARDEN JC, MORRIS JJ, SMITH TC. Pulmonary hemodynamics during general anesthesia in man. *Anesthesiology* 1969; **30**: 629–36.
8. BINDSLEV L, HEDENSTIERNA G, SANTESSON J, GOTTLIEB I, CARVALLHAS A. Ventilation-perfusion distribution during inhalation anaesthesia. Effects of spontaneous breathing mechanical ventilation and positive end-expiratory pressure. *Acta Anaesthesiologica Scandinavica* 1981; **25**: 360–71.
9. TOKICS L, HEDENSTIERNA G, STRANDBERG A, BRISMAR BO, LUNDQUIST H. Lung collapse and gas exchange during general anesthesia: effects of spontaneous breathing, muscle paralysis, and positive end-expiratory pressure. *Anesthesiology* 1987; **66**: 157–67.
10. KNIGHT RJ, HOUGHTON IT. Field experience with the Triservice anaesthetic apparatus in Oman and Northern Ireland. *Anaesthesia* 1981; **36**: 1122–7.
11. SHOULDER PJ. The management of missile injuries. *Journal of the Royal Naval Medical Service* 1983; **69**: 80–4.
12. SLATER EM, NILSSON SE, LEAKE DL, PARRY WL, LAVER MB, HEDLEY-WHITE J, BENDIXEN HH. Arterial oxygen tension measurements during nitrous oxide-oxygen anaesthesia. *Anesthesiology* 1965; **26**: 642–7.
13. NUNN JF. *Applied respiratory physiology*, 3rd edn. London: Butterworths, 1987.
14. MARTIN DG, KONG KL, LEWIS GTR. Resistance to airflow in anaesthetic breathing systems. *British Journal of Anaesthesia* 1989; **62**: 456–61.
15. AKINYEMI OO, ADELAJA AB. Blood gas studies using spontaneously respired halothane in ambient air. *Anaesthesia* 1982; **37**: 353–4.
16. MACKIE AM. Drawover anaesthetic systems. Factors determining the inspired oxygen concentration. *Anaesthesia* 1987; **42**: 299–304.
17. RAINE JM, BISHOP JM. A-a difference in  $O_2$  tension and physiological dead space in normal man. *Journal of Applied Physiology* 1963; **18**: 284–8.
18. HEWLETT AM, HULANDS GH, NUNN JF, MILLEDGE JS. Functional residual capacity during anaesthesia III: Artificial ventilation. *British Journal of Anaesthesia* 1974; **46**: 495–503.
19. MARSHALL BE, COHEN PJ, KLINGENMAIER CH, AUKBERG S. Pulmonary venous admixture before, during, and after halothane: oxygen anaesthesia in man. *Journal of Applied Physiology* 1969; **27**: 653–7.
20. DUECK R, PRUTOW RJ, DAVIES NJH, CLAUSEN JL, DAVIDSON TM. The lung volume at which shunting occurs with inhalation anaesthesia. *Anesthesiology* 1988; **69**: 854–61.
21. NUNN JF, FREEMAN J. Problems of oxygenation and oxygen transport during haemorrhage. *Anaesthesia* 1964; **19**: 206–16.

### The role of a critical care unit in an epidemic

A. Kumar, MD, DA, Lecturer, S. Gupta, MD, Senior Resident, S. Ahuja, MD, DA, Lecturer, A. Sethi, MD, DA, Assistant Professor, A. Bhattacharya, MD, DA, Professor and Head, Department of Anaesthesiology and Critical Care, University College of Medical Sciences and G.T.B. Hospital, Dilshad Garden, Delhi 110095, India.

#### Summary

*The role of a critical care unit in life-threatening situations is well established. The management of 52 children with acute gastroenteritis and 22 children with acute paralytic poliomyelitis as part of recent epidemics is described. The solutions to the problems in the critical care management of these 74 victims (out of a total of 6197 patients admitted during the epidemics) are discussed.*

#### Key Words

*Infection; poliomyelitis, gastroenteritis.  
Intensive care.*

Medical institutions all over the world often have to face mass casualties caused by natural and other causes. Some centres have developed disaster service facilities, and disaster medicine is rapidly becoming a recognised subspecialty. Anaesthetists are now considered as important members of such teams by virtue of the wide experience they have gained from critical care units. However, it is now rare for a critical care unit to become involved in the management of an infectious epidemic. In this paper we present the role of our critical care unit in the management of victims of two epidemics that ravaged parts of Delhi recently.

#### Case reports

A total of 6197 severely affected patients were received in the casualty department of our hospital over a period of 2 months. Twenty-two percent were discharged on the same day and treatment continued at their homes; the remainder were admitted for periods ranging from 1 day to 3 weeks. A total of 74 children were treated in the critical care unit. This group comprised 52 patients with acute gastroenteritis (aged 3 months–11 years) and 22 patients with acute paralytic poliomyelitis (aged 8 months–2 years).

Initially, a separate gastroenteritis ward was set up by the hospital authorities. It was realised very soon that many of these patients were critically ill and needed intensive management that was not possible in this area because of lack of space, staff and equipment. The members of the anaesthesiology department had to visit the ward frequently to resuscitate these children and it was believed that a separate critical care unit with an early resuscitation facility and follow-up in a special area would be of great help in management. A plan was drawn up and necessary arrangements were made within the existing constraints of space and staff. A three-bedded area labelled as the critical care area was commissioned adjacent to the existing four-bedded critical care unit. The number of patients overwhelmed the facilities and restricted the working of the unit on many occasions. It was often necessary to treat two children in each adult bed.

The facilities were divided into two areas: resuscitation at the casualty department and continued care at the critical care area. In the casualty department, all necessary equipment for cardiopulmonary resuscitation (CPR) was provided. Tracheal intubation was required in 49 patients and CPR was necessary in 14. The setting up of intravenous lines in 42 patients was found to be life-saving. These patients were transferred to the critical care unit as early as possible (Table 1).

The critical care unit had all the facilities for long-term ventilation and monitoring. The ventilators used were Amsterdam mark II, Bird mark 8 and a few locally made models (Bird mark 2). There were times when, because of acute shortage of ventilators, the lungs of many of these children had to be ventilated manually with the help of residents and medical students by rotation. Paediatric infusion sets and infusion pumps were also brought into use. Monitors used included electrocardiography, noninvasive blood pressure measurement, temperature, pulse oximetry and capnography whenever required. All emergency drugs were also made available. The unit was air-conditioned with window-type air-conditioners.

The nursing staff were generally drawn from the existing critical care ward and the neonatal nursery, but it was not always possible to provide one nurse per patient. The unit was managed round the clock by the residents of the department of anaesthesiology and critical care under the supervision of a consultant, with members of the paediatric department on call.

An analysis of various patients admitted to the critical

Table 1. Emergency management ( $n = 74$ ).

Procedure	Number of patients
Setting up intravenous line	42
Tracheal intubation	49
Cardiopulmonary resuscitation	14

This paper was presented at the First Critical Care Conference held in Bombay, India on 25 November 1989, at the XXXVIII National Conference of the Indian Society of Anaesthetists, held at Manipal, India on 28 December 1989 and also at the Intensive Care Workshop on 29 November 1989 held at Aiims, New Delhi, India.

Correspondence to: Dr A. Kumar III/H-14, Lajpat Nagar, New Delhi-110 024, India

Accepted 11 June 1990.

**Table 2.** Analysis of major complications in 74 patients admitted to the critical care unit.

Indication	Number of patients	Age group
Moderate to severe and severe dehydration with gross electrolyte imbalance	47	6 months–8 years
Respiratory arrest	35	6 months–4 years
Cardiorespiratory arrest	14	6 months–5 years
Peripheral circulatory failure	25	4 months–6 years
Seizures	11	5 years–8 years
Hypothermia	22	6 months–5 years

care unit is shown in Table 2. The largest group suffered severe dehydration with gross electrolyte imbalance, which was evident in 47 patients. Mechanical ventilation was required in 49 patients. Humidified oxygen was given by mask in another 25 patients.

Intravenous fluid therapy was the mainstay of treatment. Initial emergency treatment for severe dehydration comprised 20 ml/kg saline 0.9% infused over a period of 30–60 minutes. The replacement protocol for lost fluid consisted of 80–100 ml/kg saline 0.45% for moderate dehydration and 120–150 ml/kg saline 0.45% for severe dehydration. This volume was infused over 4–6 hours. The first half was given rapidly over 2 hours and the second half over the next 4 hours. Maintenance fluid was calculated as 25 ml/kg/6 hours of Isolyte P, and for on-going losses, 10 ml/kg/hour of saline 0.45% was added. The intravenous infusion was continued if the child remained unconscious or if there was persistent vomiting or paralytic ileus. Potassium chloride 20 mmol was added to each litre of intravenous fluid in cases of normokalemia and 40 mmol/litre in hypokalemia, when urine production started. As soon as the child was conscious enough and fit enough, oral rehydration solution (ORS) was given liberally.

Intermittent suction of the oropharynx and tracheal tube was carried out at regular intervals. Initially a combination of antibiotics (ampicillin and cotrimoxazole) was started. These antibiotics were changed later according to stool culture/sensitivity reports. Physiotherapy and postural drainage were undertaken whenever possible. Good nursing care contributed greatly towards the physical needs of the paralysed patients.

It was observed during follow-up that the diaphragm and intercostal muscles were involved in 18 patients with poliomyelitis. The spectrum of the paralytic disease was enormously variable and characteristically the picture was of an asymmetrical distribution. In patients with acute gastroenteritis, the haematocrit was found to be raised and estimation of serum electrolytes revealed isotonic dehydration in the majority of patients. Stool culture/sensitivity was done wherever required and revealed organisms such as *Vibrio cholerae* (commonest), *Salmonella*, *Shigella* and *E. coli* (enterotoxigenic and enteroinvasive strains). Blood urea nitrogen was found to be raised in a few patients with acute gastroenteritis.

Forty-three of the 52 patients with acute gastroenteritis

recovered and were transferred to the paediatric ward; nine other patients died. The majority of the survivors stayed in the critical care unit for 1–3 days. Twelve of the 22 patients with acute poliomyelitis died; 10 recovered and were transferred to the paediatric ward. The duration of stay of these patients in the critical care unit was 18–24 days (Table 3).

### Discussion

This epidemic situation was reminiscent of the catastrophic epidemic at Greater Copenhagen which occurred in the second half of 1952. During the Copenhagen epidemic, 2722 patients with poliomyelitis (866 with paralysis and 1856 without) were admitted over a period of 4.5 months.<sup>1,2</sup> That epidemic was instrumental in initiating the concept of critical care units. In our series the majority of 6197 patients were suffering from acute gastroenteritis and this posed a very different kind of problem, with serious derangement of metabolic function<sup>3–7</sup> culminating in cardiorespiratory failure. In many patients, it was impossible to identify the precise diagnosis at the time of admission.

Five of the nine patients with gastroenteritis who died did so because of gross electrolyte imbalance; four others were admitted at a very late stage of their illness and although they could be revived initially, they succumbed ultimately to severe metabolic derangement. There was a dramatic improvement in most other patients as soon as fluid and electrolyte losses were replaced. *Vibrio cholerae* responded best to doxycycline therapy.

Outbreaks of cholera are not uncommon in developing and tropical countries. Recent reports include one epidemic in West Africa in 1970 that caused 150 000 cases of cholera. In South Africa, 25 244 bacteriologically proved cases were reported during six epidemics of cholera.<sup>3</sup> Cholera is endemic in areas of southern and south east Asia including Bangladesh,<sup>4</sup> India,<sup>5</sup> Pakistan, Nepal, Indonesia, Singapore<sup>6</sup> and Thailand. Transmission is seasonal in some estuarine cities like Calcutta.<sup>7</sup> There are few data available regarding the role of critical care units in the management of these patients.

We were particularly unfortunate in suffering two simultaneous epidemics. A total of 104 patients with poliomyelitis (22 with paralysis and 82 without) were admitted over a period of 2 months. This number is very small compared to the Copenhagen epidemic but these patients needed much more prolonged therapy and ventilatory support compared to those with gastroenteritis.

During the 1952 epidemic, Blegdam hospital in Copenhagen had only one tank (Emerson) and six cuirass respirators. This equipment proved wholly insufficient when the epidemic developed into a major catastrophe. In the present epidemic we had four Bird (mark 8) and two Amsterdam (mark II) ventilators that also proved to be insufficient and had to be supplemented with manual bag ventilation by residents and medical students in rotation. During the 1952 epidemic, manual bag ventilation was continued for periods of up to 3 months by relays of 1400 medical students who were paid 30 shillings (Krone) per 8-hour shift.<sup>1,2</sup> Our students requested no payment. They worked in 6-hour shifts, although their services were required for only a few weeks.

In the present epidemic, delayed admission and very severe encephalitis were responsible for most of the deaths in patients with paralytic poliomyelitis. The mortality rate was highest in bulbospinal poliomyelitis (62.5% compared to 33% in the spinal poliomyelitis group). In the 1952 epidemic, early tracheostomy with insertion of a tightly fitting cuffed rubber cuff tube, repeated aspiration, postural drainage and bag ventilation reduced mortality from 80% to 40%.<sup>1,2</sup>

**Table 3.** Results.

	Acute gastroenteritis	Acute paralytic poliomyelitis
Total admissions	52	22
Survival	43 (82.7%)	10 (45.5%)
Death	9 (17.3%)	12 (54.5%)
Duration of stay in critical care unit	1–3 days	18–24 days



It is evident from these experiences that even with severe constraints regarding manpower, equipment and space, the outcome in terms of survival in these groups of children can be quite gratifying. It would have been difficult to treat such a large number of patients if we had not organised a separate critical care unit.

We believe that the survival rate would have been much higher if more of the patients could have been brought to the critical care unit. We have learnt from our experience that a state of preparedness is essential for any large institution to tackle catastrophic situations; this includes formation of a disaster committee, marking of separate areas, formation of a staff rota and assigning duties to specific individuals.

#### References

1. LASSEN HCA. A preliminary report on the 1952 epidemic of poliomyelitis in Copenhagen with special reference to the treatment of acute respiratory insufficiency. *Lancet* 1953; **1**: 37-41.
2. IBSEN B. The anaesthetist's viewpoint on the treatment of respiratory complications in poliomyelitis during the epidemic in Copenhagen, *Proceedings of the Royal Society of Medicine* 1954; **47**: 72-4.
3. KUSTNER HGV, GIBSON IHN, CARMICHAEL TR, VANZYL L, CHOULER CA, HYDE JP, DU PLESSIS JN. The spread of cholera in South Africa. *South African Medical Journal* 1981; **60**: 87-90.
4. SAMADI AR, SHAHID N, EUSOF A, YUNUS M, HUQ MI, KHAN MU, RAHMAN ASMM, FURUQUE ASG. Classical *Vibrio cholerae* biotype displaces El tor in Bangladesh. *Lancet* 1983; **1**: 805-7.
5. CHAURASIA AR. Cholera in India: 1950-80. *Journal of Indian Association for Communicable Diseases* 1983; **6**: 14-23.
6. GOH KT. Epidemiological surveillance of communicable diseases in Singapore. *Tropical Diseases Bulletin* 1985; **82**: 365-6.
7. MILLER CJ, DRASER BS, FEACHAM RG. Cholera and estuarine salinity in Calcutta and London. *Lancet* 1982; **29**: 1216-8.

*Anaesthesia*, 1991, Volume 46, pages 59-61

### Factors that influence the induction dose of propofol

G. J. McCleane, MB, BCh, FFARCSI, D. F. Fogarty, MB, BCh, FFARCSI, Registrars. C. H. Watters, MB, BCh, FFARCSI, Senior Registrar, Department of Anaesthetics, Craigavon Area Hospital, Craigavon, Co Armagh, Northern Ireland.

#### Summary

*Factors that influence the induction of anaesthesia with propofol were investigated in a prospective study of 1000 patients. Pre-operative albumin and urea concentrations correlated with the minimum induction dose of propofol, but less strong correlations were found with haemoglobin, globulin and total protein concentrations. Age was an important influence on the induction dose of propofol ( $r = -0.34$ ) which was also closely related to ASA grade. Induction of anaesthesia with propofol is dependant on a number of variables, and this study suggests that pre-operative albumin and urea concentrations are important.*

#### Key words

*Anaesthetics, intravenous; propofol.  
Induction; dose.*

The use of the intravenous induction agent propofol has become established in modern anaesthetic practice. A dose of 2.5 mg/kg achieves induction in 95% of healthy unpremedicated patients,<sup>1</sup> although most anaesthetists reduce this dose in the less fit patient. Age has a significant effect on the necessary induction dose;<sup>2</sup> elderly patients require a mean of 1.5 mg/kg of this agent to induce anaesthesia.<sup>3</sup> This may be because of alterations in the volume of distribution found in older patients, which is known to have a significant effect on the induction dose of any rapidly acting intravenous anaesthetic.<sup>4</sup> Elderly patients in addition, have a tendency to have lower cardiac outputs, hepatic and renal blood flows and total body water than younger patients,<sup>5</sup> and these factors may affect the induction dose of an intravenous anaesthetic.

Edwards and Ellis<sup>6</sup> have demonstrated that the induction dose of thiopentone correlates with the pre-operative haemoglobin level, but not with the plasma albumin, globulin or albumin-globulin ratios, while Dundee and

Hassard<sup>7</sup> confirmed the correlation to pre-operative haemoglobin levels and demonstrated an inverse relationship between the induction dose of thiopentone and the plasma urea concentration. It is therefore clear that there are a number of factors that have an influence on the induction dose of thiopentone. The importance of the results of pre-operative blood investigations in respect of the induction dose of propofol has not yet been established.

The aim of this study was to investigate the relationship between the induction dose of propofol and the pre-operative haemoglobin, urea, total protein, albumin, globulin and albumin globulin ratios, and to confirm the relationship between the induction dose of propofol and the patients age in premedicated subjects.

#### Method

One thousand patients over 14 years of age admitted for both routine and emergency surgical procedures were

studied. Each patient was allocated to an ASA grade by the anaesthetist depending on their history and results of physical examination at the pre-operative visit. Each received a temazepam premedication 1.5 hours before anaesthesia: those under 60 kg received 10 mg, those of 61 to 90 kg 20 mg, and those of over 90 kg received 30 mg.

Patients were given 1 mg/kg propofol over a 30-second period and observed for 30 seconds. The eyelash reflex was tested at the end of this time and, if present, a further 10 mg propofol was administered, and after 15 seconds the reflex tested again. This was repeated until the eyelash reflex was lost, and this dose of propofol was taken as the minimum induction dose (MID) of the agent. This method is similar to that described by Dundee and colleagues<sup>5</sup> in respect of thiopentone. All the anaesthetics were given by one of the authors.

Results of haemoglobin, urea, total protein, albumin and globulin estimations were recorded (if present) in addition to the gender, age, ASA grade and minimum induction dose of propofol. Results were analysed using correlation coefficients and regression analysis; *p* values of less than 0.05 were considered statistically significant.

### Results

The number of patients with each particular investigation are shown in Table 1, along with the mean and range of results seen with these investigations.

The minimum induction dose (MID) in females was 1.51 mg/kg (SD 0.37) and in males 1.53 mg/kg (SD 0.40). A negative correlation between the logarithm of the MID of propofol and patient age was seen and is described by the equation:

$$\log \text{propofol} = 0.26 - 0.002 \text{ age } (r = -0.34, p < 0.00001)$$

No patients in ASA grade 5 were studied. The mean MID of propofol according to ASA grade is shown in Table 2. A close relationship was found when the mean MID of propofol was compared with the ASA grade (correlation coefficient 0.99, *p* < 0.01).

Positive correlations were seen when albumin concentrations and albumin/globulin ratios were compared with the MID of propofol. A negative correlation existed between urea concentration and the MID of propofol. Globulin and haemoglobin concentrations were only weakly negatively correlated with the MID of propofol (Table 3).

### Discussion

In the 1000 patients studied a very strong correlation existed between the minimum induction dose of propofol and the ASA grade. This confirms the clinical impression that ill patients require less propofol to induce anaesthesia than those without disease. The correlation between age and MID of propofol was less strong (*r* = -0.34) and is in agreement with the results of Hilton and colleagues,<sup>9</sup> who found a correlation coefficient of -0.37 in their series. These findings support the utility of the ASA grading

**Table 2.** Mean minimum induction dose of propofol (mg/kg) according to American Society of Anesthesiologists grade.

ASA grade	Number	Mean minimum dose (mg/kg; SEM)
1	562	1.60 (0.015)
2	287	1.46 (0.020)
3	128	1.36 (0.037)
4	23	1.14 (0.072)

**Table 3.** Relationship between age, haemoglobin, urea, total protein, albumin and globulin concentrations and albumin/globulin ratio and minimum induction dose of propofol and log minimum induction dose of propofol.

Variable	Correlation coefficient (propofol)	Correlation coefficient (log propofol)
Age Males	-0.31*	-0.31*
Age Females	-0.32*	-0.36*
Haemoglobin	0.09****	0.12***
Urea	-0.24*	-0.25*
Total protein	0.12***	0.16*
Albumin	0.28*	0.27*
Globulin	-0.08*****	-0.12*****
A/G ratio	0.24**	0.25**

\* = *p* < 0.00001; \*\* = *p* < 0.0001; \*\*\* = *p* < 0.001; \*\*\*\* = *p* < 0.01; \*\*\*\*\* = *p* < 0.1.

system and emphasise the disparity between chronological age and physical health.

Albumin concentration had the greatest influence on the dose of propofol needed to induce anaesthesia. Propofol is 97–99% protein bound<sup>10</sup> and this may explain this relationship. Thiopentone, in contrast, is between 72 and 81% protein bound<sup>11,12</sup> and consequently the relationship between albumin concentration and the induction dose of thiopentone is much less marked.<sup>6</sup>

Alterations in protein binding occur in patients with renal impairment. Ghoneim and Pandya<sup>11</sup> have demonstrated that 56% of thiopentone remains unbound in patients with renal disease, compared to 28% in normal controls and this explains the sensitivity of uraemic patients to thiopentone.<sup>13</sup> A similar explanation may be advanced for the statistically significant correlation between urea concentrations and the MID of propofol.

A correlation coefficient of 0.09 was found when haemoglobin concentration was compared with the MID of propofol. Interestingly, exactly the same coefficient was found by Dundee and Hassard<sup>7</sup> when haemoglobin concentration was compared to the MID of thiopentone. It may be, in view of the differences in protein binding and pharmacokinetics of these agents, that this correlation coefficient represents the effects of systemic disease, which may

**Table 1.** Mean and range of results of pre-operative investigations.

Investigation	Number	Mean	Range
Haemoglobin g/dlitre	945	13.3	8.1–17.1
Urea mmol/litre	851	5.4	1.5–18.6
Total protein g/dlitre	851	68.5	46–88
Albumin g/dlitre	299	39.3	27–51
Globulin g/dlitre	299	30.0	18–47
Albumin/globulin ratio	299	1.33	0.77–2.45

cause anaemia, on the MID of propofol rather than a direct effect of haemoglobin concentration on the unbound cerebral propofol level which produces the desired loss of consciousness.

The MID in this study overall was 1.50 mg/kg and is considerably less than the normally recommended dose.<sup>1</sup> Three explanations may exist for this. Firstly, loss of eyelash reflex was taken as the endpoint in this study, and not infrequently limb movement occurred after loss of this reflex. Therefore, a slightly larger dose was often needed to induce anaesthesia in the conventional sense. Secondly, the studies that show a need for 2.25–2.5 mg/kg to induce anaesthesia were in unpremedicated patients,<sup>1,3,14</sup> and all patients in this study received a temazepam premedication. Thirdly, our impression is that the slow injection technique used in this study lessened the requirement of propofol.

Many factors have an influence on the induction of anaesthesia. We have demonstrated in this study the relationship between the results of routine laboratory investigations and the induction of anaesthesia with propofol. Protein binding is clearly important. However, to achieve the necessary central concentrations of induction agents to produce loss of consciousness, the distribution of these agents after administration must also be relevant. Alterations in the volume of distribution of thiopentone, associated with age, produce a decrease in dose required to induce anaesthesia.<sup>15</sup> Similarly, changes in the pharmacokinetics with ageing have an effect on the onset time of midazolam.<sup>16</sup> It is likely that any process that alters the pharmacokinetics of a drug would also influence the necessary induction dose of propofol. Such processes include disease states and anxiety, the latter of which leads to catecholamine release<sup>17</sup> and alterations in peripheral blood flow. Central tolerance to the depressant effects of induction agents may also occur and explain the increased requirements of these agents in patients with alcohol addiction.

## References

1. CUMMINGS GC, DIXON J, KAY NH, WINDSOR JPW, MAJOR E, MORGAN M, SEAR JW, SPENCE AA, STEPHENSON DK. Dose requirements of ICI 35,868 (Propofol, 'Diprivan') in a new formulation for induction of anaesthesia. *Anaesthesia* 1984; **39**: 1168–71.
2. ROBINSON FP, DUNDEE JW, HALLIDAY NJ. Age affects the induction dose of propofol ('Diprivan'). *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 157–9.
3. MCCOLLUM JSC, DUNDEE JW, HALLIDAY NJ, CLARK RSJ. Dose response studies with propofol ('Diprivan') in unpremedicated patients. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 85–7.
4. STANSKI DR, WATKINS WD. *Drug disposition in anaesthesia*. New York: Grune and Stratton, 1982.
5. LEITHE ME, HERMILLER JB, MAGORIEN RD, UNVERFERTH DV, LEIER CV. The effect of age on central and regional hemodynamics. *Gerontology* 1984; **30**: 240–6.
6. EDWARDS R, ELLIS FR. Clinical significance of thiopentone binding to haemoglobin and plasma protein. *British Journal of Anaesthesia* 1973; **45**: 891–3.
7. DUNDEE JW, HASSARD TH. The influence of haemoglobin and plasma urea levels on the induction dose of thiopentone. *Anaesthesia* 1983; **38**: 26–8.
8. DUNDEE JW, HASSARD TH, MCGOWAN WAW, HENSHAW J. The 'induction' dose of thiopentone. A method of study and preliminary illustrative results. *Anaesthesia* 1982; **37**: 1176–84.
9. HILTON P, DEV VJ, MAJOR E. Intravenous anaesthesia with propofol and alfentanil. The influence of age and weight. *Anaesthesia* 1986; **41**: 640–3.
10. KIRKPATRICK T, COCKSHOTT ID, DOUGLAS EJ, NIMMO WS. Pharmacokinetics of propofol (Diprivan) in elderly patients. *British Journal of Anaesthesia* 1988; **60**: 146–50.
11. GHONEIM MM, PANDYA H. Plasma protein binding of thiopental in patients with impaired renal or hepatic function. *Anesthesiology* 1975; **42**: 545–9.
12. MARK LC, PEREL JM, BRAND L, DAYTON PC. Studies with thiohexital, an anesthetic barbiturate metabolized with unusual rapidity in man. *Anesthesiology* 1968; **29**: 1159–66.
13. DUNDEE JW, RICHARDS RK. Effect of azotemia upon the action of intravenous barbiturates and anesthesia. *Anesthesiology* 1954; **15**: 333–46.
14. DUNDEE JW, ROBINSON FP, MCCOLLUM JSC, PATTERSON CC. Sensitivity to propofol in the elderly. *Anaesthesia* 1986; **41**: 482–5.
15. HOMER TD, STANSKI DR. The effect of increasing age on thiopental disposition and anesthetic requirement. *Anesthesiology* 1985; **62**: 714–24.
16. HALLIDAY NJ, DUNDEE JW, LOUGHRAN PG, HARPER KW. Age and plasma proteins influence the action of midazolam. *Anesthesiology* 1984; **61**: A357.
17. FELL D, DERBYSHIRE DR, MAILE CJD, LARSSON IM, ELLIS R, ACHOLA KJ, SMITH G. Measurement of plasma catecholamine concentrations. An assessment of anxiety *British Journal of Anaesthesia* 1985; **57**: 770–4.

### Awareness during Caesarean section

G. Lyons, FFARCS, R. Macdonald, PhD, FFARCS, Consultants, Department of Anaesthesia, St James' University Hospital, Leeds LS9 7TF.

#### Summary

*Between 1982 and 1989 over 3000 patients were questioned about recall and dreaming after general anaesthesia for Caesarean section. Some 28 (0.9%) patients were able to recall something of their operation and 189 (6.1%) reported dreams. There was uniform adherence to a rigid anaesthetic protocol up to and including 1985, but a much publicised incident reported from the courtroom stimulated a relaxation of this regimen. Consequently the incidence of awareness decreased from 1.3% to 0.4%, and the incidence of dreaming was also reduced. Recollections of surgery were confined to manipulations, noises and voices. None of our patients complained of pain at the time of interview, although one since has. The inadequacies of the initial protocol and an approach to informed consent are discussed.*

#### Key words

Anaesthesia; obstetric.  
Complications; awareness.

The technique of general anaesthesia for Caesarean section described by Moir<sup>1</sup> became popular during the 1970s. The incidence of awareness associated with this technique, less than 1%,<sup>2</sup> tended to be regarded as an acceptable complication. One particular incident in 1985 was followed through the courts by the national press,<sup>3</sup> and as a result patients were alerted to the possibility of this complication, and obstetric anaesthetists had to recognise another potential source of litigation. It was suggested in a recent case report<sup>4</sup> that all patients in the maternity unit should be visited after general anaesthesia, to both elicit awareness and arrange sympathetic counselling when it was appropriate. It was also suggested that strict obligatory protocols for general anaesthesia be avoided.

All patients who receive general anaesthesia for Caesarean section in St James' University Hospital, Leeds, are visited by an anaesthetist as part of a general post-operative review. They are asked during the course of the interview 'Do you remember anything about your operation?' If the answer is negative, they are asked 'Did you dream during your operation?', and if appropriate the content of the dream is explored. Records of these post-operative reviews exist from 1982 to the present. The unit has moved away from a rigid protocol to a more flexible approach during the 8 years between 1982 and 1989. The influence of this on the incidence of awareness together with a clinical portrait drawn from the experiences of 8 years of continuous audit are presented below.

#### Incidence of awareness

During the 8 years under review some 3076 general anaesthetics were required for Caesarean section and 28 patients (0.9%) were able to recall something of their operation. A further 189 (6.1%) had dreams. The patient was deemed to be aware if the content of the dream represented unequivocal recall of an event that took place in the operating theatre. The incidence of awareness and dreaming is shown in Table 1. Approximately 5% of patients were lost to follow up, and a smaller percentage did not speak English.

Our protocol for general anaesthesia up to and including 1985 was as follows: induction, thiopentone 3-4 mg/kg, suxamethonium 100 mg to facilitate tracheal intubation, a

Table 1. General anaesthesia for Caesarean section. (Incidence of awareness and dreaming).

Year	Number	Aware	Dreamt
1982	434	3 (0.7%)	28 (6.4%)
1983	548	7 (1.3%)	47 (8.6%)
1984	323	6 (1.8%)	22 (6.8%)
1985	345	6 (1.7%)	25 (7.2%)
1986	385	2 (0.5%)	24 (6.2%)
1987	344	1 (0.3%)	13 (3.8%)
1988	404	3 (0.7%)	19 (4.7%)
1989	293	0	11 (3.7%)

therapeutic dose of a nondepolarising muscle relaxant, and maintenance with 50% oxygen/nitrous oxide with 0.5% halothane. The gas concentrations were changed after delivery to 30% oxygen in nitrous oxide, and papaveretum 0.3 mg/kg (or equivalent) was given intravenously. The halothane was turned off after delivery of the baby.

This protocol was significantly relaxed during 1986, and as a result the induction dose of thiopentone increased to 5 to 7 mg/kg. Isoflurane 1% replaced halothane 0.5% and was frequently continued until the end of the operation at a lower concentration.

The overall incidence of awareness was 1.3% up to and including 1985, and since then it has been 0.4%. The incidence of dreaming was 7.4% and 4.7% respectively over the same period.

#### Patients' experiences

No patient, at the time of interview, was able to recall the entire operation or claimed to have experienced pain, although one since has, some 3 years after the event. Her claim was not supported by the answers she gave at post-operative interview. Recollections were limited to intubation, surgical manipulations, crying babies and other theatre noises.

One patient was distressed by her experience:

*Case report 1.* A 26-year-old multipara required Caesarean section for failed induction. The epidural sited for labour was not used and she was given a general anaesthetic at 0300 hours. Induction was according to the

rigid protocol, with thiopentone 250 mg. No mention is made of volatile agent, but almost certainly halothane 0.5% was used. Pethidine 100 mg was given at delivery. The record of her postoperative review reads as follows: 'Delayed going to sleep. Remembers induction, not being able to move and choking in throat and going to theatre and voices, then nothing'.

The same patient required emergency Caesarean section for cord prolapse and suspected scar dehiscence one year later. The trainee anaesthetist was acquainted with her previous history but had no time to seek advice. The anaesthetic given was identical to the first except that papaveretum 20 mg was given at delivery. The patient complained of awareness in the immediate postoperative period.

The patient was anxious to discuss her experience at review and was upset and tearful. She asked to visit the operating theatre so that she might understand what had happened to her, and this was arranged the same day. The record reads as follows: 'Visit to anaesthetic room and operating theatre: patient shown all equipment and described what would have happened to her—patient can describe accurately what happened in terms of what was said and sensations e.g. remembers leg falling off table while being moved into theatre. Can remember until after baby delivered and gas concs changed. Very anxious before going to O.T. and anaesthetic room (found anaesthetic room frightening) but very relieved by being shown everything. Found sounds disturbing e.g. ventilator, Dinamap, sucker and was glad to know what they were. Will visit again tomorrow'. The record of the following day reads: 'Patient much happier today. Doesn't think she will be afraid of a GA next time'.

Not all patients respond negatively to the experience:

*Case report 2.* A woman made an appointment to discuss her recollections of a Caesarean section carried out at another hospital. She convincingly described the entire course of the operation and said she had experienced pain. The details of the general anaesthetic were unavailable. She heard God speak to her during induction. She had earned a small income as an astrologist before her operation, but as a result of this experience she gave up astrology and became an Evangelical Christian. When asked if her life had been changed for better or worse, she replied 'For the better'.

Unless the content of dreams is probed a significant number of cases will go unreported:

*Case report 3.* An anaesthetic nurse denied any recollection of her operation but admitted to dreaming. She was unable to say whether her dream was good or bad and described it as 'abstract'. She said, when pressed, the only part of it she could recall was the word 'Fraser'. This was readily traced back to a conversation that had occurred during the operation concerning the sudden death of Gillian Fraser's mare.

The commonest form of dream reported is of friends and relatives in pubs and discotheques, and one woman dreamt that she was taking part in a television programme called 'Game For a Laugh'. However, even this form of dream may amount to unenlightened recall:

*Case report 4.* A woman denied any recollection of her operation, but described dreaming of a happy gathering of friends and relatives in a pub. She commented on a 'clicking noise' in the background, and then went on spontaneously to mimic the unmistakable sound of a Manley ventilator.

### Discussion

The technique for general anaesthesia described by Moir in 1970<sup>1</sup> has become the standard for Caesarean section. His

recommendation for halothane 0.5% rather than 0.8% has been widely adopted, and no patients in his study were aware, but since then the technique has been acknowledged to have an incidence of awareness under 1%.<sup>2</sup> Crawford found that awareness was abolished with halothane 0.5%, but was 1.6% with halothane 0.4%.<sup>5</sup> The output of some halothane vaporizers, including the Fluotec Mark 3, at a setting of 0.5%, has been shown to vary from 0.34% to 1.06%.<sup>6</sup> It would seem, therefore, that many halothane vaporizers are not sufficiently accurate to work within the constraints set by Moir and that even minor variations in output might be responsible for an unacceptable degree of awareness. The use of 0.5% halothane for Caesarean section should be abandoned unless accurate vapour monitoring is available and all vaporizers used for obstetric patients should be serviced regularly.

The inadequacies of a rigid protocol are revealed in the story of the woman who was aware twice. Isoflurane replaced halothane because the manufacturers were prepared to give us free vaporizers as part of a marketing drive. We have not subjected our babies to neonatal scoring systems, but we are able to say that our neonatologist has not appreciated that our vapour has changed, or that the mothers are receiving much larger doses of thiopentone. Our impression is that abandoning the rigid protocol has had no detectable impact on the condition of the babies born.

The overall incidence of awareness was 1.3% up to 1985, whilst the protocol was applied, and since then it has been 0.4%. It should be recognised that the major impetus to change was the fear of litigation, and the realisation that our aware patients represented a sizable reservoir of medico-legal liability.

Our experience suggests that unless patients are questioned specifically about recall during surgery significant under-reporting is likely to occur. Offset against this is the difficulty of distinguishing between true recall and emergence phenomena. This is impossible if the recollections are of, for example, crying babies and manipulations. It is even possible that pain experienced during turning at the end of the operation could be misinterpreted. Crawford<sup>5</sup> believed that unpleasant dreams and awareness are associated and that 10% of aware patients feel pain. Our experience supports neither of these contentions, and all our patients have been pain free. The incidence of dreaming since 1985 has declined from 7.4% to 4.7%, and our impression is that the content of the dreams has become less lurid.

Only one of our 28 aware patients has sought the services of a solicitor. Perhaps our patients do not regard their experiences as being other than trivial complications. It seems that an opportunity to discuss any problem with an anaesthetist postoperatively is all the counselling that is required.

The nature of informed consent for Caesarean section under general anaesthesia is a topic for debate. It would seem appropriate to acquaint the patient with the risks if she is in a position to exercise a choice between general and regional anaesthesia. This involves informing the patient that there is a less than 1:100 chance of recall, but pain will not be part of the experience. This seems to be acceptable to most women without causing undue anxiety. If the patient has no choice of anaesthetic technique, unless there is a specific request to discuss risks, this will serve no purpose other than increasing her level of anxiety.

Three patients were aware in 1988: one was shocked, some might consider this a mitigating circumstance.<sup>7</sup> One remembered closure, this was avoidable. One remembered tracheal intubation despite thiopentone 400 mg, this was barely credible. Efforts to reduce the incidence of awareness have been worthwhile, but we do not yet feel sufficiently

confident to be able to guarantee the abolition of recall.

#### Acknowledgments

We are grateful to our junior staff who over the years have given most of the general anaesthetics and have assisted in the postoperative reviews.

#### References

1. MOIR DD. Anaesthesia for Caesarean section. An evaluation of a method using low concentrations of halothane and 50 per cent of oxygen. *British Journal of Anaesthesia* 1970; **42**: 136-42.
2. MOIR DD, THORNBURN J. *Obstetric anaesthesia and analgesia*, 3rd edn. Eastbourne: Baillière Tindall, 1986.
3. *Daily Telegraph* 1985, June 8.
4. BRAHAMS D. Caesarean section: pain and awareness without negligence. *Anaesthesia* 1990; **45**: 161-2.
5. CRAWFORD JS. The principles and practice of obstetric anaesthesia, 5th edn. Oxford: Blackwell, 1985.
6. ROSENBERG PH, ALILA A. Accumulation of thymol in halothane vaporisers. *Anaesthesia* 1984; **39**: 581-3.
7. CRAWFORD JS. Fetal well being and maternal awareness. *British Journal of Anaesthesia* 1988; **61**: 247-8.

*Anaesthesia*, 1991, Volume 46, pages 64-67

### Coagulation screening before epidural analgesia in pre-eclampsia

P. Barker, FCAnaes, Registrar, C.C. Callander, FFARCS, Senior Registrar, Department of Anaesthetics, University Hospital of Wales, Cardiff, UK.

#### Summary

A questionnaire survey of current practice at a small cross-section of obstetric units, covering 22% of all United Kingdom deliveries, revealed a marked lack of standard practice regarding requests for coagulation screens on pre-eclamptic patients who require epidural procedures. A retrospective audit was therefore carried out on 434 coagulation screens requested for pre-eclamptic patients in whom epidural analgesia might have been considered. Borderline abnormalities of coagulation were found in only 10 patients (2%). Platelet counts of less than  $150 \times 10^9/\text{litre}$  were present in 28% of cases. 'Significant' thrombocytopenia ( $< 100 \times 10^9/\text{litre}$ ) and all coagulation abnormalities were only encountered in severe pre-eclampsia (diastolic blood pressure of greater than 110 mmHg and proteinuria of ++ or greater). Furthermore, coagulation abnormality was always associated with a reduced platelet count (mean,  $97 \times 10^9/\text{litre}$ ). This study would therefore support anaesthetic practice which restricted any requests for coagulation testing to severe pre-eclamptic patients only. For these patients first line testing could be limited to a platelet count.

#### Key words

Anaesthetic techniques, regional; epidural.  
Complications; pre-eclampsia, coagulopathies.  
Anaesthesia; obstetric.

Disordered coagulation, whether through abnormal platelet function and number or disseminated intravascular coagulation is an integral part of pre-eclamptic toxæmia (PET).<sup>1</sup> In anaesthetic practice any coagulopathy is considered a contraindication to epidural or spinal anaesthetic procedures because of the dangers of epidural haematoma formation leading to spinal cord compression and irreversible damage. For this reason it is common practice to carry out full coagulation screening before epidural or spinal procedures for patients with PET. The impression gained from such patients in this centre was that whilst the platelet count was not infrequently reduced, it was very rare to encounter abnormal coagulation results from the standard laboratory tests of prothrombin time, thrombin clotting time, kaolin cephalin clotting time and fibrinogen titre.

It was therefore decided to carry out a survey of current practice at a cross-section of United Kingdom obstetric units and thereafter a detailed review of the results from coagulation screens of patients with PET in the two Cardiff maternity units.

#### Methods

The initial component of this study consisted of a postal survey of current practice at a limited cross-section of maternity units throughout Great Britain and Northern Ireland. The units selected to receive questionnaires were those at which a designated consultant obstetric anaesthetist could be easily identified. Forty-three questionnaires were sent. Details were sought about the number of annual deliveries and practice concerning platelet/coagulation testing before epidurals for patients with PET. A range of tests were listed and the question asked: are any of these tests used and if so are they carried out always on every case of PET, only if severe PET and only if indicated by first line tests.

The second component of this study consisted of a retrospective audit on all available coagulation screens for such patients at the two Cardiff maternity units. All results of coagulation screens are stored at the coagulation laboratory and these were exhaustively reviewed so as to extract



**Table 1.** Summary of all available coagulation results carried out on pre-eclamptic patients before delivery at University Hospital of Wales, (mean number of annual deliveries 2700; mean epidural rate 20%) and St Davids Hospital, Cardiff (mean number of annual deliveries 2800; mean epidural rate 22%). Period covered, 1973–1989.

Patients with normal platelet count and normal coagulation	312 (72%)
Patients with platelets $100\text{--}150 \times 10^9/\text{litre}$ but normal coagulation	79 (18%)
Patients with platelets $<100 \times 10^9/\text{litre}$ and normal coagulation	33 (8%)
Patients with platelets $>150 \times 10^9/\text{litre}$ but abnormal coagulation	1*
Patients with platelets $100\text{--}150 \times 10^9/\text{litre}$ but abnormal coagulation	4 (1%)
Patients with platelets $<100 \times 10^9/\text{litre}$ and abnormal coagulation	5 (1%)
Total number of coagulation screens	434

\*Platelet count in this patient was  $153 \times 10^9/\text{litre}$ .

all tests on obstetric patients. These results were then cross-matched with obstetric records to exclude any patients tested for reasons other than PET, such as antepartum haemorrhage, intra-uterine death, anticoagulant therapy and any other nonPET related coagulopathy. Furthermore, tests were only included if they were carried out at a stage of pregnancy when epidural or spinal procedures were or might have been considered i.e. cases in whom delivery was in prospect. Postnatal results were excluded.

Results from one unit (University Hospital of Wales, Cardiff) were available from 1973 to 1989 and from the second unit (St Davids Hospital, Cardiff) from 1985 to 1989. During the period of the retrospective survey it was anaesthetic practice for all patients with PET, regardless of severity, to have a platelet count and coagulation screen (one stage prothrombin time, thrombin clotting time, kaolin cephalin clotting time and fibrinogen titre) carried out before any epidural or spinal procedure.

Abnormal coagulation (local laboratory definition) was taken as a kaolin cephalin clotting time (KCCT) ratio of patient to control of greater than 1.28 i.e. greater than the ratio of 45 seconds to 35 seconds, or an isolated patient time of greater than 50 seconds. Similarly, thrombin clotting time (TCT) and one stage prothrombin time (OSPT) ratios of greater than 1.28 patient: control were taken as abnormal, as were fibrinogen titres of less than 1/64. Case notes of all patients with PET with abnormal coagulation results were extracted so as to record any anaesthetic procedures carried out and the severity of the PET. Severe

PET was taken as a diastolic blood pressure of 110 mmHg and proteinuria ++ or greater on dip-stick testing.<sup>2</sup>

An abnormal platelet count was taken as less than  $150 \times 10^9/\text{litre}$  since this is the normal haematological definition of thrombocytopaenia. However, details of PET severity and anaesthetic procedures were only extracted from case records of those patients with a count of less than  $100 \times 10^9/\text{litre}$ , since this is the level below which epidural administration is usually said to be contraindicated.<sup>3</sup>

### Results

Table 1 summarises the results of platelet counts and coagulation tests carried out on PET patients in whom delivery was in prospect. A full breakdown of the coagulation results for the 10 patients with abnormal coagulation is shown in Table 2, together with details of those patients in this group who had suffered an eclamptic fit and those who had had epidural or general anaesthesia administered.

All 10 patients in the abnormal coagulation group had severe PET, and nine had a platelet count of less than  $150 \times 10^9/\text{litre}$ ; the remaining case had a platelet count of  $153 \times 10^9/\text{litre}$ .

Platelet counts of less than  $100 \times 10^9/\text{litre}$  occurred in 38 patients i.e. 9% of all cases, all 38 had severe PET. The mean platelet count for all PET cases was  $199 \times 10^9/\text{litre}$  (SEM 3.901). For patients with normal coagulation tests the mean count was  $219 \times 10^9/\text{litre}$  (SEM 4.628) whilst that for cases with abnormal coagulation was  $97 \times 10^9/\text{litre}$  (SEM 11.688).

Table 3 gives results of the questionnaire survey into current practice across the United Kingdom. Thirty-seven consultant anaesthetists responded, a response rate of 86%. The size of the units varied from 1–7000 annual deliveries (mean 4100). A total of approximately 271 United Kingdom obstetric units are recognised by the Royal College of Obstetricians and Gynaecologists for training purposes and therefore this survey only covered 14% of such units.<sup>4</sup> However, the number of annual deliveries at the units in the survey amounts to 22% of the national total of approximately 680 000.<sup>4</sup>

Eight units had policies restricting requests for platelet counts and coagulation tests to severe cases of PET only. In one unit fibrin degradation product levels were requested on all severe PET patients whilst platelet count testing was reserved as a second-line test. Two respondents stated that bleeding times were not routinely used but would be for any patients taking aspirin.

### Discussion

Clear guidelines on coagulation screening for PET cases before epidural or spinal procedures will reduce unne-

**Table 2.** Details of abnormal coagulation tests and patient details (\*abnormal test results). Patients 9 and 10 had an eclamptic fit before delivery; patients 2 and 5 had epidurals administered; patients 1, 3, 4, 6, 8 and 10 had general anaesthetics.

Patient	Platelet count	Fibrinogen titre	TCT (patient time seconds/control time seconds)	OSPT	KCCT
1	153	1/128	14/13	15/15	50/37*
2	147	1/128	14.5/13	12/13	55/37*
3	114	1/128	19.5/14.5*	12/13.5	40/39
4	109	1/128	14/13	13/13	50/37*
5	102	1/128	11.9/11.4	11.4/12.5	51.4/41*
6	86	1/64	17/12*	16/13	46/37
7	84	1/128	16.5/12*	10/12.5	50/47
8	83	1/128	20/13*	21/12.5*	51.5/36*
9	62	1/8*	17.5/12*	19/12*	43/36
10	30	1/128	15.5/13.5	16.5/13.5	51/47*

**Table 3.** Results of national questionnaire about practice when ordering coagulation screens for pre-eclampsics before epidurals. Total number of obstetric units responding to questionnaire, 37.

Test	Percentage of units using test prior to epidurals on pre-eclamptic patients			
	Always on every case of PET	Only if 'severe' PET	Only if indicated by first line tests	Test very rarely or never used for PET cases
Platelet count	73%	24%	3%	—
Prothrombin time	51%	37%	6%	6%
Thrombin clotting time	35%	34%	6%	25%
KCCT	27%	3%	—	70%
Fibrinogen titre	22%	27%	30%	21%
FDPs	9%	43%	23%	25%
Bleeding time	9%	18%	18%	55%
Hemochron system	—	—	9%	91%

cessary testing. This will help to reduce costs and will allow more prompt institution of such procedures for many patients with PET. In centres where all these patients are screened, the fact that at least one hour's delay may be incurred whilst coagulation results are awaited will result in some needlessly foregoing the benefits of regional analgesia or anaesthesia. The objective of this study was to assess the need for such coagulation tests.

The results of the questionnaire survey reveal great variation in current practice that ranges from full coagulation screening of all patients with PET to only limited testing e.g. only platelet count or prothrombin time and only for severe PET. This lack of standard practice possibly reflects the relative absence of published studies directly addressing this question as it relates to anaesthetic management. The results of this study may help to establish correct or at least more rational practice.

This was not a review of all PET cases or a survey of PET at any specific stage of pregnancy, unlike many previous studies in this area.<sup>5</sup> The patients included were purely those in whom delivery was imminent and who had or might have presented to anaesthetists. The PET patients included therefore form a group which has not previously been studied but which is of great clinical relevance to anaesthetists, obstetricians and haematologists. The number of patients included in this group is much less than the total number of PET cases during the period of the survey, because many patients did not have coagulation tests requested since no epidural or spinal procedure was planned and because of the exclusion of patients with associated problems.

The importance of the severity of PET with regard to the occurrence of coagulation abnormalities has been previously suggested.<sup>1,6</sup> However, this is the first large study to show an absolute restriction of coagulopathy to only severe cases. This may justify the limiting of coagulation screens to only severe cases of PET, but needless to say any patient who has clinical signs of a bleeding diathesis should have a full and immediate coagulation screen.

The results of this study also show that all cases with a platelet count of less than  $100 \times 10^9/\text{litre}$  had severe PET. This level is usually taken as the cut-off point below which epidural procedures are not advised,<sup>3</sup> so this finding may support restriction of platelet count testing as well as coagulation screening to only severe cases. It is of note that the results of the questionnaire survey show that many centres are already restricting all screening tests to severe cases. One consideration with regard to the possible restriction of platelet count testing to only severe PET is the concern that cases of the rare HELLP syndrome (haemolysis, elevated liver enzymes and low platelet count) may be missed. A low platelet count can be encountered with this

condition despite otherwise mild signs of PET.<sup>7</sup> However, it has been suggested that this syndrome is actually a separate condition from PET<sup>8</sup> and furthermore it will always be possible to encounter unexpected profound thrombocytopaenia from non-pregnancy related conditions.<sup>9</sup>

A further finding was that all the PET patients with abnormal coagulation results had a markedly reduced platelet count (mean  $97 \times 10^9/\text{litre}$ , SEM 11.688). It might therefore be reasonable to use a platelet count as a single first line test for all patients with PET to be screened, but the level of platelet count below which full coagulation screening would be indicated is not clear. One patient (number 1) had a slightly prolonged KCCT with a platelet count of  $153 \times 10^9/\text{litre}$ . This count is higher than the normal point at which thrombocytopaenia is defined i.e.  $150 \times 10^9/\text{litre}$ . Therefore it may be prudent to carry out a full coagulation screen on all severe cases of PET with a platelet count of less than  $200 \times 10^9/\text{litre}$ , although it must be admitted that this is a relatively arbitrary cut-off point. The questionnaire responses again revealed that certain centres are already restricting screening tests in a manner which the results of this study would justify i.e. using the platelet count as the sole first line test. However, in comments added by certain respondents it would appear that counts above  $100 \times 10^9/\text{litre}$  are often taken as confirming the absence of coagulopathy. The results of this study suggest that this may not be so in severe PET, although it is worth noting that none of the coagulation derangements seen in this study were very marked.

The questionnaire survey revealed wide variation in the type of tests used for coagulation screening. The most commonly used test other than platelet count was the prothrombin time and often this was the only test carried out. In this study an abnormal KCCT was often the only test suggesting deranged coagulation. It would therefore seem reasonable to always include this latter test in any screen.

A further test which is at present rarely used is the bleeding time. It has often been pointed out that one of the fundamental problems in PET is platelet malfunction even when the actual count is normal or only moderately reduced.<sup>10,11</sup> Tests of platelet function such as the bleeding time would therefore seem to be the most appropriate of all, but for various reasons its use has not been widely recommended in the past,<sup>12,13</sup> although this situation may change with the increasing use of aspirin for PET prophylaxis.

In conclusion, it is obvious that no single study, however large, could hope to identify, exactly, those patients with PET who are at risk of an epidural haematoma. This is even more apparent when one considers the fact that the levels of thrombocytopenia and disseminated intravascular

coagulation at which the risk becomes clinically relevant are totally unknown. The results of this study, despite these limitations, may help to rationalise anaesthetic practice with regard to requests for coagulation screens on such patients.

#### Acknowledgments

We thank Professor A.L. Bloom, Haematology Department, University Hospital of Wales and Dr G.A.D. Rees, Consultant in Anaesthesia, University Hospital of Wales for their constructive criticisms of early drafts of this paper.

#### References

1. HOWIE PW, PRENTICE CRM, McNICOL GP. Coagulation, fibrinolysis and platelet function in pre-eclampsia, essential hypertension and placental insufficiency. *Journal of Obstetrics and Gynaecology of the British Commonwealth* 1971; **78**: 992–1003.
2. CUNNINGHAM FG, MACDONALD PC, GANT NF. Hypertensive disorders in Pregnancy. In: CUNNINGHAM FG, MACDONALD PC, GANT NF, eds. *Williams obstetrics*. London: Prentice Hall International, 1989: 653–94.
3. CRAWFORD JS. Intensive care of the severely hypertensive woman. In: SHARP F, SYMONDS EM, eds. *Hypertension in pregnancy: proceedings of the sixteenth study group of the Royal College of Obstetricians and Gynaecologists*. New York: Perinatology Press, 1987: 345.
4. HIBBARD BM, SCOTT DB. The availability of epidural anaesthesia and analgesia in obstetrics. *British Journal of Obstetrics and Gynaecology* 1989; **97**: 402–5.
5. THORNTON JG, MOLLOY BJ, VINALL PS, PHILIPS PR, HUGHES R, DAVIES JA. A. prospective study of haemostatic tests at 28 weeks gestation as predictors of pre-eclampsia and growth retardation. *Thrombosis and Haemostasis* 1989; **61**: 243–5.
6. EVANS SR. Anesthesia for parturients with toxemia of pregnancy. In: DATTA S, OSTHEIMER GW, eds. *Common problems in obstetric anesthesia*. Chicago: Year Book Medical, 1987: 315–22.
7. DUFFY BL. HELLP syndrome and the anaesthetist. *Anaesthesia* 1988; **43**: 223–5.
8. GOODLIN RC. Expanded toxemia syndrome or gestosis. *American Journal of Obstetrics and Gynecology* 1986; **154**: 1227–33.
9. HEW-WING P, ROLBIN SH, HEW E, AMATO D. Epidural anaesthesia and thrombocytopenia. *Anaesthesia* 1989; **44**: 775–7.
10. WHIGHAM KAE, HOWIE PW, DRUMMOND AH, PRENTICE CRM. Abnormal platelet function in pre-eclampsia. *British Journal of Obstetrics and Gynaecology* 1978; **85**: 28–32.
11. RAMANATHAN J, SIBAI BM, VU T, CHAUHAN D. Correlation between bleeding times and platelet counts in women with preeclampsia undergoing Cesarean section. *Anesthesiology* 1989; **71**: 188–91.
12. LETZKY EA. *Coagulation problems during pregnancy*. Edinburgh: Churchill Livingstone, 1985: 13.
13. ROLBIN SH, ABBOTT D, MUSCLOW E, PAPSIN F, LIE LM, FREEDMAN J. Epidural anesthesia in pregnant patients with low platelet counts. *Obstetrics and Gynecology*. 1988; **71**: 918–20.

*Anaesthesia*, 1991, Volume 46, pages 67–69

### Modification of pain on injection of propofol—a comparison between lignocaine and procaine

M. E. Nicol,\* MB, ChB, DA, FFARCS, Registrar, J. Moriarty,† MB, ChB, BAO, FFARCSI, Senior Registrar, J. Edwards, FFARCS, D. S. Robbie, FFARCS, Consultants, R. P. A'Hern, MSc, Statistician, The Royal Marsden Hospital, Fulham Road, London SW3 6JJ.

#### Summary

*Pain on injection of propofol was assessed in a controlled, randomised study of 273 patients. They received either lignocaine 10 mg, procaine 10 mg or isotonic saline 0.5 ml, 15 seconds before the injection of propofol into a vein on the back of the hand. The incidence of pain on injection in the control group (51%) was comparable with other studies. Lignocaine and procaine both significantly reduced the pain (35% and 34% respectively,  $p < 0.05$ ) but there was no statistical difference between these two groups.*

#### Key words

*Anaesthetics, intravenous; propofol.  
Analgesics; local; lignocaine, procaine.  
Complication; pain.*

The use of intravenous lignocaine to reduce the pain on injection of propofol has been well documented.<sup>1–3</sup> Procaine, injected intravenously, is known to improve the flow of intravenous infusions,<sup>4</sup> and also reduces the pain on injection of propofol.<sup>5</sup> The present study was carried out to compare the efficacy of these two local anaesthetics in reducing the incidence and severity of the pain associated with propofol injection.

#### Methods

A total of 383 ASA 1–2 patients aged 15–86 years were allocated at random to receive either 0.5 ml isotonic saline, 0.5 ml 2% lignocaine or 0.5 ml 2% procaine. Exclusion criteria were pregnancy, any patient requiring rapid sequence induction, patients in whom a difficult intubation was anticipated and those with any history of reaction to

\* Current appointment: Senior Registrar, National Hospital for Nervous Diseases, Queen's Square, London.

† Current appointment: Senior Registrar, St James' Hospital, Dublin 8.

Accepted 1 June 1990.

local anaesthetic agents. The study had the approval of the Hospital Ethics Committee.

The premedication was left to the discretion of the anaesthetist and consisted of either intramuscular papaveretum and hyoscine, oral temazepam or no premedication.

A 23-gauge Y-Can (Wallace UK Ltd) was inserted into a vein on the back of the hand on arrival in the anaesthetic room. Lignocaine 10 mg, procaine 10 mg or isotonic saline was injected over 2 seconds. The local anaesthetic agents were not mixed with the propofol, in order to comply with the manufacturers published recommendations, but were given as separate injections. This was followed 15 seconds later by 2.5 mg/kg propofol given over 30 seconds.

The patient was asked, after 10 seconds of this latter injection, if the arm was comfortable and any pain experienced was graded by the assessor as mild, moderate, or severe. Anaesthesia was maintained with oxygen, nitrous oxide and a volatile agent. Fentanyl and muscle relaxants were used as required. Patients were again questioned immediately before their discharge from the recovery ward. They were asked if the injection that had put them to sleep had been painful and, if so, to grade the pain as mild, moderate or severe.

The pH of the agents used was measured using a Corning 140 pH meter calibrated against buffers of known pH.

Results were analysed using Mann-Whitney trend and Chi-squared tests. McNemars test was used to compare pain felt in the anaesthetic room with that remembered in the recovery room.

### Results

Table 1 shows the numbers, ages and weights of the patients in each group. The difference in numbers between the groups occurred because more operations in the procaine group were cancelled after randomisation than in the other two groups. However, a Chi-squared value of 1.05 shows this to be not significant.

**Pain.** The results of the assessment of pain in the anaesthetic room are shown in Table 2. The overall incidence of pain was 51% in the control group, 35% in the lignocaine group and 34% in the procaine group. The incidence of severe pain was least in those who had received procaine (5%). This is statistically significant ( $p < 0.05$ ) compared with the control group (15%), but is not so in comparison with the lignocaine group (13% incidence of severe pain).

**Premedication.** Oral temazepam was given to 74 patients in the control group, 77 in the lignocaine group and 70 in the procaine group. No premedication was given to 18, 13 and 11 patients in each group respectively. The number of

**Table 1.** Demographic data. Values are expressed as mean (SD).

	Control ( <i>n</i> = 95)	Lignocaine ( <i>n</i> = 95)	Procaine ( <i>n</i> = 83)
Age; years	50 (19–86)	52 (18–85)	52 (15–83)
Weight; kg	65 (46–109)	65 (43–123)	63 (39–101)

**Table 2.** Assessment of pain in the anaesthetic room.

Allocation	<i>n</i>	Pain			
		None	Mild	Moderate	Severe
Control	95	47	18	16	14
Lignocaine	95	62	10	11	12
Procaine	83	55	12	12	4

**Table 3.** A comparison of pain felt in the anaesthetic room with that remembered in the recovery room.

	Pain in recovery room		
	Pain	No pain	Total
Patients with pain in anaesthetic room	17	92	109
Patients with no pain in anaesthetic room	2	162	164
Total	19	254	—

patients given an intramuscular premedication was too small to be considered separately, thus a comparison was made between the incidence of painful injection in those patients who were given oral premedication with those who were given none. There was no statistical difference between the incidence of pain on injection whether the patients had received an oral premedication or not.

**Pain felt vs pain remembered.** The results in Table 3 show that, whereas 109 patients (40%) had pain in the anaesthetic room, only 19 patients (7%) remembered the injection as painful ( $p < 0.001$ ).

**pH of agents.** The agents used had the following mean pH values: lignocaine 5.10 (SD 0.17), procaine 4.16 (SD 0.64), isotonic saline 5.21 (SD 0.10) and propofol 8.00 (SD 0.15).

### Discussion

The principal finding of this study was that there was no statistical difference between procaine and lignocaine in their ability to reduce the pain on injection of propofol through small peripheral veins.

The mechanism of pain on injection is not fully understood, but the activation of kininogens has been suggested.<sup>6</sup> Pain is felt in the forearm some 15 to 20 seconds after the start of the injection when dorsal hand veins are used. It is interesting that very small amounts of local anaesthetic given a short time before, can affect the incidence of pain at a more proximal site when the agent can neither be presumed to have reached that site nor have had time to bind to and influence nerve conduction. Local anaesthetics have direct effects on vascular smooth muscle which may influence their own systemic absorption and duration of action. Lignocaine has been used to prevent pain on injection with other induction agents<sup>7,8</sup> when administered by varying methods. We are unaware of any study evaluating the use of procaine in this respect. Procaine is an ester linked local anaesthetic. It has a lower pH than lignocaine and therefore less free base is available for rapid penetration and it has a slower onset of action. However, a shorter duration of action<sup>9</sup> coupled with low toxicity,<sup>10</sup> warranted its investigation. Propofol, when diluted to a concentration of 5 mg/ml, has been shown to reduce the pain on injection into hand veins<sup>11</sup> whereas injection into a fast running infusion does not help.

The use of large veins markedly reduces the frequency of pain on injection of propofol,<sup>12</sup> but the veins on the dorsum of the hand have the advantage of being readily accessible, of minimising the risk of inadvertent intra-arterial injection and extravasation is easy to see. The use of oral premedication made no difference to the incidence of pain felt at induction, in agreement with a previous study.<sup>13</sup>

Few patients who felt pain at induction remembered pain when questioned in the recovery room. This is in sharp contrast with a previous study<sup>12</sup> and therefore warrants further investigation.

In conclusion, procaine is as effective as lignocaine in

reducing the pain on injection of propofol into small hand veins.

### References

1. HELBO-HANSEN S, WESTERGAARD V, KROGH BL, SVENDSEN HP. The reduction of pain on injection of propofol: the effect of the addition of lignocaine. *Acta Anaesthesiologica Scandinavica* 1988; **32**: 502-4.
2. McCULLOCH MJ, LEES NW. Assessment and modification of pain on induction with propofol (Diprivan). *Anaesthesia* 1985; **40**: 1117-20.
3. BROOKER J, HULL CJ, STAFFORD M. Effect of lignocaine on pain caused by propofol injection. *Anaesthesia* 1985; **40**: 91-2.
4. ORGANE GSW, SCURR CF. Intravenous procaine in transfusion. *British Medical Journal* 1948; **2**: 787.
5. ROBBIE DS. Anaesthesia for surgery in malignant disease of the head and neck. In: FILSHIE J, ROBBIE DS, eds. *Anaesthesia and malignant disease*. Edward Arnold, 1989: 138.
6. SCOTT RPF, SAUNDERS DA, NORMAN J. Propofol: clinical strategies for preventing the pain of injection. *Anaesthesia* 1988; **43**: 492-5.
7. MILLAR JM, MARSHALL BARR A. The prevention of pain on injection. A study of the effect of intravenous lignocaine before methohexitone. *Anaesthesia* 1981; **36**: 878-80.
8. BROWN PM, MOSS E. Reduction of pain on injection of etomidate. *Anaesthesia* 1981; **36**: 814-16.
9. COUSINS MJ, MATHER LE. Clinical pharmacology of local anaesthetics. *Anaesthesia and Intensive Care* 1980; **8**: 257-77.
10. RITCHIE JM. Mechanisms of action of local anaesthetic agents and biotoxins. *British Journal of Anaesthesia* 1975; **47**: 191-8.
11. STOKES DN, ROBSON N, HUTTON P. Effect of diluting propofol on the incidence of pain on injection and venous sequelae. *British Journal of Anaesthesia* 1989; **62**: 202-3.
12. CUMMINGS GC, DIXON J, KAY NH, WINDSOR JPW, MAJOR E, MORGAN M, SEAR JW, SPENCE AA, STEPHENSON DK. Dose requirements of 1C1 35 868 (propofol, 'Diprivan') in a new formulation for induction of anaesthesia. *Anaesthesia* 1984; **39**: 1168-71.
13. BRIGGS LP, WHITE M. The effects of premedication on anaesthesia with propofol ('Diprivan'). *Postgraduate Medical Journal* 1985; **61**(Suppl. 3): 35-7.

## Correspondence

- |   |    |  |  |
|---|----|--|--|
| <b>Supplementary oxygen and the laryngeal mask airway</b>                 | 70 | <b>Epidural catheterisation</b>  |  |
| <i>R.P. Lewis, FFARCS and M. Porter, MB, ChB</i>                          |    | <i>S.P. Ambesh, A. Kumar, I. Bhukal and H. Singh</i>                       |  |
| <b>Laryngeal mask airway and laryngeal spasm</b>                          | 71 | <b>Subcutaneous morphine</b>   |  |
| <i>M.Z. Michel and J.F. Stubbing</i>                                      |    | <i>T.J. Semple, FFARCS and P.E. Macintyre, FFARCS</i>                      |  |
| <b>ABGs as easy as ABC</b>  | 71 | <b>The pump that gave too much: accidental overinfusion of prostacylin</b> |  |
| <i>M.G. Serpell, FCAnaes</i>  |    | <i>J. Dunne, FFARCS and C. Wise, FFARCS</i>                                |  |
| <b>Withdrawal of levorphanol</b>  | 71 | <b>Accidental intra-arterial injection of tubocurarine</b>                 |  |
| <i>R.H. James, FFARCS</i>   |    | <i>E. Devlin, FFARCSI and I. Bali, FFARCSI</i>                             |  |
| <b>Intravenous cannulae colour coding</b>                                 | 72 | <b>Persistent erection and general anaesthesia</b>                         |  |
| <i>R.S. Mathias, MD</i>   |    | <i>H.E. Hosie, FFARCS and J.G. Todd, FFARCS</i>                            |  |
| <b>Successful difficult intubation</b>                                    | 72 | <b>Hazards from homely anaesthetic rooms</b>                               |  |
| <i>R. Marks, FFARCS</i>   |    | <i>R.H. James, FFARCS</i>  |  |
| <i>S. Dogra, MB, BS, R. Falconer FFARCS and I.P. Latto, FFARCS</i>        |    | <b>Fenem CO<sub>2</sub> detector device</b>                                |  |
| <b>Acid aspiration prophylaxis</b>  | 73 | <i>J.R. Brimacombe, FCAnaes</i>  |  |
| <i>J. Evans, FCAnaes, C.C. Rout, FFARCS and D.A. Roche, MRCP, FFA(SA)</i> |    |  |  |
| <b>A problem with thiopentone solution</b>                                | 74 |  |  |
| <i>M.A. Thickett, FFARCSI</i>   |    |  |  |

### Supplementary oxygen and the laryngeal mask airway

We would like to compliment Drs Broadway and Royle (*Anaesthesia* 1990; 45: 792-3) on their method of administering oxygen to patients with a Brain laryngeal mask airway *in situ* in the recovery area. We have also experienced difficulties with the use of ordinary disposable oxygen masks and have modified a Bain coaxial system for use in our recovery room. The valve was removed and the inner tubing, carrying fresh gas, was connected directly to an oxygen flowmeter. The outer tubing was cut to 30 cm (this has a volume of 120 ml) and is a convenient length so that the recovery nurse can place her hand near the end to confirm the presence of exhaled gas. The system thus acts as a T-piece with a reservoir limb of 120 ml.

This system is in routine use and has proved very useful; it is inexpensive and easy to use. The inspired oxygen concentration will depend on the fresh gas flow and on the patient's tidal volume and respiratory rate.

We measured the delivered inspired oxygen in six patients (Table 1), and also the inspired and expired carbon dioxide and respiratory rate using a Datex multinec and the tidal volume using a Wright's respirometer, at oxygen flows of 2, 4, 6 and 8 litres/minute. The tidal volumes ranged from 250 to 500 ml and respiratory rate from 6-20

Table 1.

Oxygen flow litres/second	Inspired oxygen % median and (range)
8	62 (60-90)
6	56 (50-76)
4	43 (38-70)
2	32 (31-52)

breaths/minute. The inspired carbon dioxide level was : in all cases and the expired carbon dioxide was in the range of 4.5-6 kPa. A fresh gas flow of 4 litres/minute would therefore result in an inspired oxygen concentration approximately 40% and should be adequate for most patients. Any patient with respiratory depression or consequent low tidal volume will receive a higher inspired oxygen concentration.

Peterborough District Hospital,  
Peterborough PE3 6DA

R.P. LEWIS  
M. PORTER

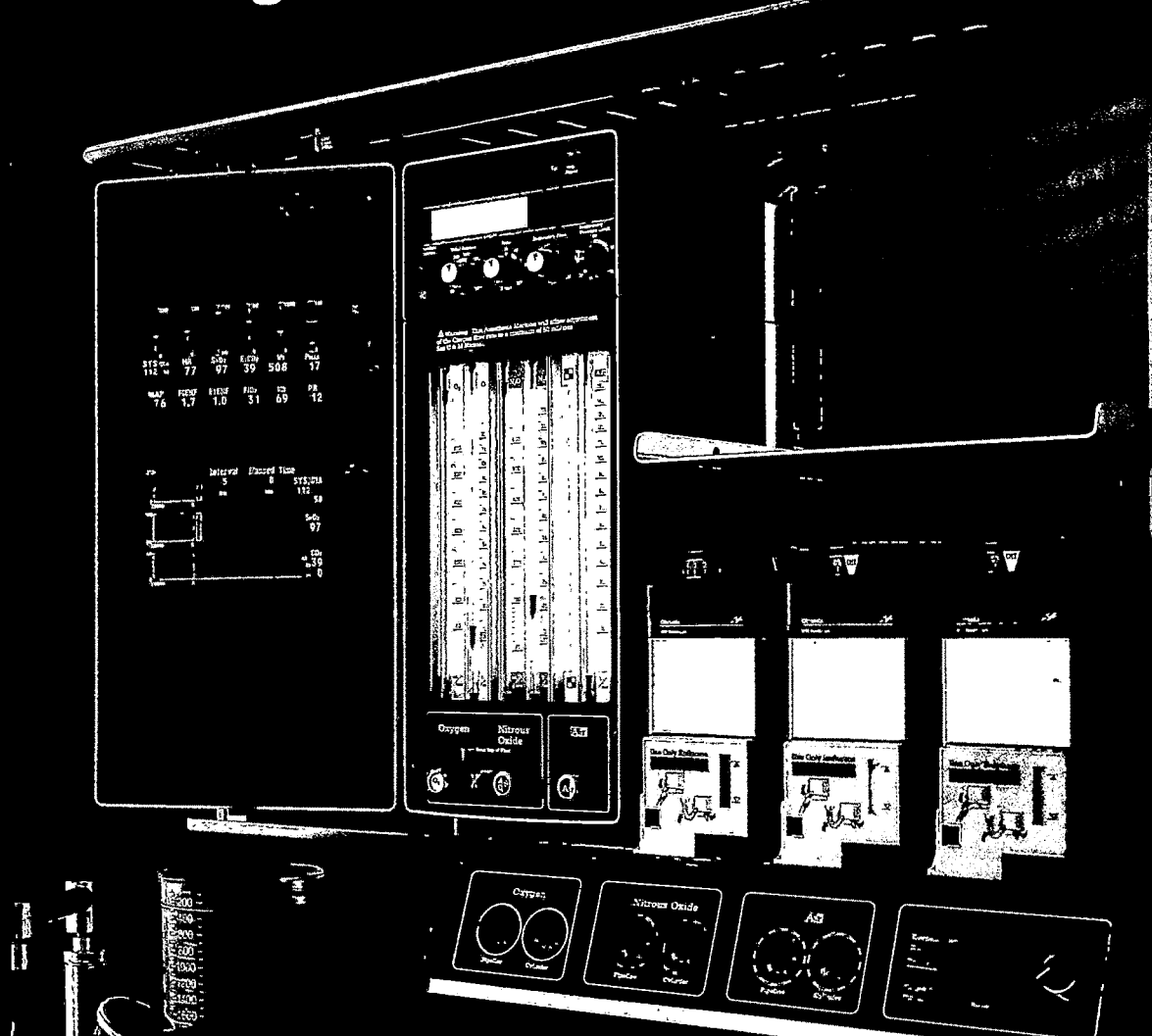
All correspondence should be addressed to Dr M. Morgan, Editor of *Anaesthesia*, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, United Kingdom.

Letters must be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters contained in the Notice to Contributors to *Anaesthesia* printed at the back of each issue. The degrees and diplomas of each author must be given in a covering letter personally signed by all the authors.

Correspondence presented in any other style or format may be the subject of considerable delay and may be returned to the author for revision. If the letter comments on a published article in *Anaesthesia*, please send three copies; otherwise two copies of your letter will suffice.



# The first true choice in sophisticated data management.



## The Modulus CD Anaesthesia System

It is another example of setting the precedent in anaesthesia: a computer-based, fully integrated anaesthesia system. More than merely acquiring data, the Modulus CD actually processes it for new depth in patient perspectives. This unique computerisation is based on the latest microprocessor concepts for the sophistication critical to effective patient and data management. This same computerisation also means that economical software upgrades can be used to keep your system up to date for years. And to help you easily use all this capability, there are no complex menus; just convenient selections when you start a case. It's simple. Reassuring. Direct.

Ergonomically organised, Modulus CD displays are equally direct. Selectable formats, waveforms and trends help you respond to data quickly. You are even warned if data drifts beyond user-set ranges, with added visualisation through a unique display that *geometrically* defines patient change as it happens.

Responsive to your technique now. Ready for your needs tomorrow. The Modulus CD is the first choice in patient care and data management. Contact an Ohmeda representative for more details.

**Ohmeda. A world of choice in anaesthesia.**

# Ohmeda

Ohmeda  
71 Great North Road  
Hatfield Hertfordshire  
England AL9 5EN  
0707 263570



**A BOC Health Care  
Company**

# Senior Medical & Health Officers (Anaesthesia)

Hospital Services Department, Hong Kong

Up to £35,600\* p.a. + 25% gratuity



- Low tax area – maximum 15% • Generous leave • Medical and dental benefits
- Accommodation allowance • Free passages and holiday visits for children
- Children's education allowance

#### THE ROLE:

Practise anaesthesia in all branches of surgery, including pre-operative and post-operative cases; manage patients in the intensive care unit; and train staff.

#### QUALIFICATIONS:

Applicants must hold (a) a United Kingdom or Irish Diploma or a recognised Commonwealth Diploma and have obtained full registration with the General Medical Council (UK); and (b) FFARCS, FFARCS (Eng or Ire), or FFA(SA); and (c) 5 years' post-registration experience.

#### TERMS OF APPOINTMENT & SALARY:

The appointment will be for an agreement of 1, 2 or 3 years. The salary scale is HK\$38,625 to HK\$44,505 per month (Approx. £30,900 to £35,604\* p.a.)

For further information and an application form write to the Hong Kong Government Office, 6 Grafton Street, London W1X 3LB, quoting reference APPT/HSD-2/90/SMHO(A). Closing date for acceptance of completed application form is 26 January 1991.

\*Based on exchange rate HK\$15 = £1 (Subject to fluctuation).

Hong Kong Government

## THE EUROPEAN ACADEMY OF ANAESTHESIOLOGY



### THE EUROPEAN DIPLOMA IN ANAESTHESIOLOGY AND INTENSIVE CARE

The 1991 Part I (MCQ) Examination will be held on

**Thursday, 26 September**

in Strasbourg, Oslo, Barcelona, Rome, Uppsala, Lund and Berne.

The Examination Regulations and Calendar, giving the criteria for entry, fees and other details, are available in English, French, German, Italian and Spanish and may be obtained from:

The Examination Secretary,  
European Academy of Anaesthesiology,  
9 Bedford Square, London WC1B 3RA, UK.  
Tel: 071 631 1650 Fax: 071 631 4352.

### Laryngeal mask airway and laryngeal apasm

The laryngeal mask airway (LMA) has been used effectively in securing the airway in cases of difficult intubation,<sup>1</sup> but there is some concern that it may provoke laryngeal spasm.<sup>2</sup> However, we used the LMA to treat laryngeal spasm after tracheal extubation in a patient who had been difficult to intubate.

An obese, 35-year-old man who was a heavy smoker presented for radical excision of pilonidal sinus. Difficult tracheal intubation was anticipated on pre-operative assessment, he had a receding jaw, short neck and his mouth opening was limited to 27 mm. It was only possible to visualise the soft palate with the mouth maximally open and the tongue maximally protruded. The patient was premedicated with papaveretum 20 mg and hyoscine 0.4 mg intramuscularly. Adequacy of the airway was assessed after pre-oxygenation and thiopentone 300 mg intravenously, and although it was difficult to maintain an adequate fit with the facemask, ventilation was possible with a high fresh gas flow. Tracheal intubation proved to be difficult, but was achieved with the help of a gum elastic bougie. Anaesthesia was then maintained with 33% oxygen in nitrous oxide, isoflurane, fentanyl 100 µg and vecuronium 8 mg. At the end of surgery he was positioned in the left lateral head-down position and neostigmine plus glycopyrronium were administered. The pharynx was sucked out under direct vision and 100% oxygen given. The trachea was extubated when the patient was breathing regularly and coughing on the tracheal tube. Following extubation he developed severe laryngeal spasm. Oxygen 100% was delivered under pressure via the facemask, but because of his receding jaw it was not possible to achieve a seal and the arterial oxygen saturation (SpO<sub>2</sub>) began to decrease. We considered a trial of an LMA worthwhile. In view of the previously difficult intubation a size 4 laryngeal mask was passed without difficulty and the cuff inflated with 30 ml of air. The patient was connected immediately

to 100% oxygen and continuous positive pressure and intermittent assisted manual ventilation could be applied with minimal leak. Although the spasm did not break, the SpO<sub>2</sub> increased over the next 20 seconds. Two minutes later the spasm was broken and the patient recovered.

Laryngeal spasm is a recognised complication after tracheal extubation, but there are several therapeutic manoeuvres that can be used to relieve it.<sup>3</sup> In this case we were unable to apply adequate positive airway pressure because the patient's receding jaw made an airtight fit with the facemask impossible. The administration of sux-methonium at this stage could have been potentially hazardous. Furthermore, what we knew to be a very difficult intubation would now be occurring in an hypoxic patient in the head-down left lateral position. Failure to intubate the trachea would have exposed him to more hypoxia and cricothyroidotomy would have been the only alternative. In spite of his limited mouth opening a size 4 LMA was inserted easily. Inflation of the cuff provided a good seal and we were able to give 100% oxygen with positive pressure without delay. The LMA may be useful in the management of laryngospasm where a tight fitting seal cannot be obtained with a facemask.

Southampton General Hospital,  
Southampton SO9 4XY

M.Z. MICHEL  
J.F. STUBBING

### References

1. BRAIN AIJ. Three cases of difficult intubation overcome by the laryngeal mask airway. *Anaesthesia* 1985; **40**: 353-5.
2. BRODRICK PM, WEBSTER NR, NUNN JF. The laryngeal mask airway. A study of 100 patients during spontaneous breathing. *Anaesthesia* 1989; **44**: 238-42.
3. ROY WL, LERMAN J. Laryngospasm in paediatric anaesthesia. *Canadian Journal of Anaesthesia* 1988; **35**: 93-8.

### ABGs as easy as ABCs

Drs Hope and Farling recommend a very useful method for converting pH to [H<sup>+</sup>] concentration (*Anaesthesia* 1990; **45**: 699). I would suggest that there is an even more simple technique. [H<sup>+</sup>] is expressed in nmol/litre and can be derived readily from the pH by remembering that a pH value of 7.40 equals a [H<sup>+</sup>] of 40 nmol/litre, and that each 0.01 unit deviation in pH corresponds to a deviation in [H<sup>+</sup>] of 1 nmol/litre in the opposite direction. For example, a change in pH from 7.45 (0.05 units more *alkaline* than normal) to 7.30 (0.10 units more *acid* than normal) is accompanied by a rise in [H<sup>+</sup>] from 35 to 50 nmol/litre.

This computation is accurate within the pH range of

7.28-7.45 and can easily be used to convert from one unit to the other and vice versa, even under examination conditions!

Ninewells Hospital,  
Dundee DD1 9SY

M.G. SERPELL

### Reference

1. KASSIRER JP, BLEICH HL. Rapid estimation of plasma carbon dioxide tension from pH and total carbon dioxide content. *New England Journal of Medicine* 1965; **272**: 1067-8.

### Withdrawal of levorphanol

Another drug has now been withdrawn from our armamentarium. So many drugs have disappeared in recent months that perhaps our response has been deadened, but withdrawal of this drug would seem to raise even more questions than usual. Studies involving levorphanol have been few, but those that have been done showed it to be much more potent than morphine with a longer action, and an incidence of vomiting and respiratory repression which was probably a little less. Certainly those of us who have used the drug have found it excellent.

Why should such a drug fail? Firstly, I should like to suggest, conservatism. The commonest postoperative analgesic prescribed is still crude poppy extract (i.e. papaveretum) even though no benefit has been shown compared with pure morphine. What chance does a later usurper stand? Secondly, the interest in postoperative analgesia of recent years has come too late. Eventually somebody would have given levorphanol by the intrathecal or extradural route or by some even less common delivery system. Perhaps it was better, as a manufacturer of a low

profit margin drug, to get it off the shelves before that occurred. Thirdly, the financing of research. University departments are being pressurised to obtain outside funds for their universities. Decisions as to which drugs will be investigated must therefore be swayed in favour of those whose manufacturers are prepared to pay to establish their safety or efficacy. What chance then for the aged, out-of-patent warhorse? Fourthly, a disproportionate belief in the importance of scientific journals! If, for the previous reasons, research is not being done it cannot be published. The drug therefore loses credibility in people's minds because they wish to be up-to-date and yet this drug never features in journals. Junior staff, the consultants of

tomorrow, only see the drug being used by consultants who cannot quote a paper in the last 10 years to support it. The downhill cycle is complete.

Unless we wish to see anaesthesia limited to a very small number of core drugs, we must learn from the withdrawal of levorphanol. No drug with a 'small, established niche' in practice is safe. Either we change the patent laws, or we tell people not to believe journals, or we persuade all who use less popular drugs to demonstrate their value at regular intervals, using the latest investigatory methods.

Leicester Royal Infirmary  
Leicester LE1 5WW

R.H. JAMES

### Intravenous cannulae colour coding

Drs Tordoff and Sweeney (*Anaesthesia* 1990; 45: 399–400) report that there is no standard colour coding on intravenous cannulae in the United Kingdom at present. An organisation called the Disposable Hypodermic and Allied Equipment Manufacturers Association of Europe, (DHAEMAE) has drafted a standard, but this is quite complex. Tordoff and Sweeney have devised a colour code that is clearer and more simple. I agree with them that it is necessary to have a national or international colour code for intravenous cannulae and that it would be very easy for the manufacturers to comply with this.

We have tried to do this in Brazil and the manufacturers agreed to change their colours to a national standard.

However, when the committee working on the draft measured the size of the cannulae, it was found that the actual sizes of the cannulae varied between manufacturers and often did not compare with the size indicated. I would like to suggest to Drs Tordoff and Sweeney that they measure the cannulae sizes of the nine manufacturers in the United Kingdom and compare them, to verify that, for example the 22 G produced by one manufacturer corresponds to the 22 G produced by another.

Al Campinas 139/41,  
01404, Sao Paulo SP,  
Brazil

R.S. MATHIAS

### Successful difficult intubation

I was interested to read the report by Dogra *et al.* (*Anaesthesia* 1990; 45: 774–6) which describes how the use of a bougie during intubation can be made easier if the tracheal tube is rotated before intubation.

The 'simulated difficult intubation' has become established for training purposes and should ideally be used by all anaesthetists in practice from time to time to maintain standards. The failure rate using a bougie without rotating the tube, however, seems depressingly high. In a study<sup>2</sup> on relatively inexperienced American residents a failure rate of 25% was noted (five failures in 20 intubations); this was using a Portex tube introduced in the normal manner under direct vision. The comparable group in the study by Dogra *et al.* showed a failure rate of 52% (13 failures in 25 intubations). This high failure rate may be attributable to an important detail of the technique.

The original technique using a bougie as described by Macintosh uses an Oxford tube. This tube has a bevel facing posteriorly,<sup>3</sup> which would help direct the tube through the cords. The advantages of the bevel on the Oxford tube were discussed by Cormack and Lehane<sup>1</sup> who argued that rotating a tube with a left facing bevel to ease it into the larynx was potentially traumatic. He advocated that the Oxford tube should always be used in this situation. However, contemporary disposable tubes all have a bevel facing to the left<sup>4</sup> and since this is now specified by the American ANSI standard it is unlikely that tubes with a posterior bevel will be manufactured in the future.

It would be advisable for Anaesthetic Departments who still possess Oxford tubes to store some in the 'difficult intubation' box.

73 Calabria Road,  
London N5 1HX

R.J. MARKS

### References

1. CORMACK RS, LEHANE J. Difficult tracheal intubation in obstetrics. *Anaesthesia* 1984; 39: 1105–11.
2. GOLDBERG JS, BERNARD AC, MARKS RJ, SLADEN RN. Simulation technique for difficult intubation: teaching tool or new hazard. *Journal of Clinical Anesthesia* 1990; 2: 21–6.
3. ALSOP AF. Non-kinking endotracheal tubes. *Anaesthesia* 1955; 10: 401.
4. BERNHARD WN, YOST L, TURNDORF H, DANZIGER F. Cuffed tracheal tubes—physical and behavioral characteristics. *Anesthesia and Analgesia* 1982; 61: 36–41.

### A reply

We welcome this opportunity to reply to Dr R.J. Marks's letter. The failure rates between our study (*Anaesthesia* 1990; 45: 774–6) and that of Goldberg *et al.*<sup>1</sup> are not comparable. Goldberg *et al.* used a simulated difficult intubation technique to pass both the gum elastic bougie and the tracheal tube, and quoted their oesophageal intubation rate. In our study we passed the gum elastic bougie under direct vision. Failure of intubation was thought to have occurred if there was a hold-up to passage of the tracheal tube over the gum elastic bougie, with only one attempt permitted and using only gentle force. The success rate would have been higher had we persisted and used more force.

The use of the gum elastic bougie was first described by Sir Robert Macintosh, but he was disappointed that in his era it was apparently not widely used. To quote an extract from a private letter (1978) from Sir Robert to Dr R.S. Cormack (with his kind permission)—'I have found the introducer to be of the greatest help, particularly abroad, when intubating in difficult (sometimes impossible) circumstances. I can honestly say that armed with an introducer I



#### Prescribing information

**Indications and dosage:** Systemic candidiasis: 400mg on the first day followed by 200-400mg once daily. Cryptococcosis, including meningitis: 400mg on the first day followed by 200-400mg once daily. Maintenance therapy to prevent relapse of cryptococcal meningitis in patients with AIDS: at least 100mg daily. Oropharyngeal candidiasis: 100-150mg once daily for 7-14 days or longer in immunocompromised patients. Other mucosal candidal infections: 50-100mg once daily for 14-30 days. Vaginal candidiasis: single 150mg dose. Use in the elderly as above except for those renally impaired - see data sheet.

**Use in children** - not recommended.

**Administration:** Diflucan may be administered either orally or by intravenous infusion at a rate of approximately 5-10ml/min. The dosages for the two routes are equivalent. **Contra-indications:** Hypersensitivity to fluconazole or related triazoles, pregnancy and women of childbearing potential unless adequate contraception is employed.

**Warnings:** Lactation: Not recommended. Renal impairment: dosage reduction may be necessary, see data sheet. **Drug interactions:** Monitor patients on concurrent anticoagulants, oral sulphonylureas, phenytoin or rifampicin. **Side-effects:** Nausea, abdominal discomfort, diarrhoea and flatulence. **Package Quantities and basic NHS Cost:** 50mg capsule, calendar pack of 7, £16.61 (PL 57/0289); 200mg capsule, calendar pack of 7, £66.42 (PL 57/0317); 150mg capsule, pack of 1, £7.12 (PL 57/0290); Bottles of 25ml and 100ml containing Diflucan 2mg/ml intravenous infusion - 25ml (50mg) bottle, £7.32; 100ml (200mg) bottle, £29.28 (PL 57/0315). Hospital prices are available on request.

**References:** 1 - 4 Data on file, Pfizer Ltd. Further information on request.



Pfizer Limited Sandwich, Kent

# Antifungal therapy that's severe on the infection, not on the patient.

Diflucan Oral/I.V. is a highly effective, well tolerated therapy with a convenient, once a day dosage and administration.

Overall clinical responses in deep-seated fungal infections are impressive: 87% in systemic candidiasis<sup>1</sup>, 96% in mucosal candidiasis<sup>2</sup> and 62% in cryptococcal meningitis.<sup>3</sup> Tolerability is similarly impressive with adverse effects comparable to placebo.<sup>4</sup>

Diflucan Oral/I.V. Treating invasive fungal infection with an iron fist and patients with a velvet glove.

**Diflucan** ORAL/I.V.  
fluconazole \*Trade Mark  
Counters invasive fungal infection



# SUBSCRIPTION ORDER FORM



**Anaesthesia** ISSN 003-2409

☐ Please enter my subscription to Volume 46, 12 issues (1991)  
Full Rate: £132.00 (UK only)/U.S. \$244.00 (Overseas)

☐ Please send me a free sample copy, including *'Instructions to Authors'*

## Method of Payment

☐ Cheque/Money order enclosed (payable to 'Harcourt Brace Jovanovich Ltd.')

☐ Purchase order enclosed      ☐ Please invoice me

☐ Credit Card

American Express/Visa (Barclaycard)/Diners Club/MasterCard (Please delete as appropriate)

Card Number \_\_\_\_\_ Expiry Date \_\_\_\_\_

Name \_\_\_\_\_

Address \_\_\_\_\_

Signature \_\_\_\_\_ Date \_\_\_\_\_

(Your signature is essential especially when paying by credit card)

## ARE YOU GETTING THE INFORMATION YOU NEED ?

Please tick the appropriate boxes below and return this questionnaire whether or not you choose to order at this time.

☐ Please continue mailing me information on Academic Press publications.

I have completed my mailing address above.

☐ Please remove my name from your mailing list.

I have completed my mailing address above.

☐ Please change my mailing address as I have indicated above.

☐ I would prefer to stay informed about these particular areas only:

Please photocopy this form and return it to:

Journals Marketing Department  
Harcourt Brace Jovanovich Ltd.  
24-28 Oval Road, London NW1 7DX, UK

or to:

Journals Production Department  
Academic Press Inc.  
1250 Sixth Avenue, San Diego, CA 92101, USA

have never failed, provided I have been able to see the back of an arytenoid. I tried for years to persuade manufacturers (e.g. Portex) to make sets of introducers of say, six different sizes, but to no avail. The answer has always been that there is no demand. To me it is clear that young anaesthetists have never been taught the value of an introducer in difficult cases.<sup>7</sup>

It appears that the frequency of use of the gum elastic bougie is gradually increasing and it is now probably the most popular method used by anaesthetists in the UK to facilitate a difficult intubation. However, we believe that the optimum size, composition and flexibility of introducers have not yet been established and further work should serve to define this. Eschmann<sup>2</sup> are now producing gum elastic bougies in a range of gauges from paediatric to large adult size.

We agree with Drs Cormack and Lehane<sup>3</sup> that rotating a tracheal tube to ease it into the larynx is potentially traumatic, and we urge that, as in our study, the tube should be withdrawn away from the cords prior to rotation. Consequent to the reduction in availability of Oxford tubes, most clinicians have only limited experience

with them, and these tubes have their own inherent difficulties and problems. For these reasons we suggest it may be better to use Magill pattern tubes but to rotate them on the gum elastic bougie. Portex<sup>4</sup> have, at last, kindly agreed to produce a trial batch of tracheal tubes with posterior facing bevels. The introduction of these into clinical practice may, however, be some years away.

Anaesthetic Department,  
University Hospital of Wales,  
Cardiff CF4 4XW

S. DOGRA  
R. FALCONER  
I.P. LATTO

#### References

1. GOLDBERG JS, BERNARD AC, MARKS RJ, SLADEN RN. Simulation technique for difficult intubation: teaching tool or new hazard. *Journal of Clinical Anesthesia* 1990; 2: 21-6.
2. ESCHMANN LTD., Peter Road, Lancing, W. Sussex BN15 8TJ.
3. CORMACK JS, LEHANE J. Difficult tracheal intubation in obstetrics. *Anaesthesia* 1984; 39: 1105-11.
4. Portex Ltd., Hythe, Kent CT21 6JL.

#### Acid aspiration prophylaxis

Dr Ng Winton and colleagues (*Anaesthesia* 1990; 45: 436-8) set out to determine whether a single oral dose of the new proton pump inhibitor omeprazole 40 mg was effective in increasing the pH of gastric residue above 2.5 at the time of anaesthetic induction. Whilst their results indicate that they achieved their aim the subsequent statement that such a regimen decreased significantly the number of patients at risk of serious pulmonary damage is premature and we are concerned that widespread use of this new drug will ensue.

Acid aspiration syndrome is a potentially life threatening complication, albeit with an extremely low incidence in relation to the actual numbers of anaesthetics administered, but its severity is well recognised to be a function of both the pH and the volume of the gastric juice that is aspirated. In 1974 Roberts and Shirley arbitrarily defined the patient at risk as someone with at least 25 ml of gastric juice at a pH less than 2.5 at the time of induction.<sup>1</sup> This statement was made on the basis of one experiment in a single monkey, which remained unpublished until 1980.<sup>2</sup> Under this definition the accepted safe threshold for pH is 2.5. However, acid aspiration pneumonitis has been reported following aspiration of gastric contents at pH 3.5<sup>3</sup> and we agree with Ng Winton *et al.* that the additional safety margin is justified.

The role of volume is less clear. The experiment by Roberts and Shirley involved unilateral pulmonary instillation of 0.4 ml/kg gastric acid and they extrapolated this to represent 25 ml in a 70-kg human adult. The critical volume of aspirate has recently been studied in a monkey model where it was shown that volumes up to 0.6 ml at pH 1 did not cause severe pneumonitis.<sup>4</sup> In the monkey model it would appear that critical volume is nearer 0.8 ml/kg, which would represent 50 ml if extrapolated to humans. To define a patient to be at risk one must consider both pH and volume. When one re-examines the paper by Ng Winton *et al.* under currently accepted conditions (pH < 3.5, volume > 25 ml) only one patient is at risk in the control group and none are at risk in the study group. With

the small number of patients studied, neither clinical nor statistical conclusions can be made regarding the influence of omeprazole on the incidence of patients at risk. At present, prophylaxis against acid aspiration is provided in our obstetric unit by the single pre-induction administration of 30 ml 0.3 M sodium citrate. With this regimen and using the above definition, approximately 2 to 6% of our patients are at risk at induction. To prove that the addition of another agent would significantly decrease the percentage of patients at risk would require a minimum of 600 patients.<sup>5</sup>

In summary, we are concerned that widespread pre-operative therapy with omeprazole would have considerable financial costs without proven benefit and that the latter will only be demonstrated when the number of patients at risk of aspiration pneumonitis are significantly reduced.

University of Natal,  
PO Box 17039  
Congella 4013  
South Africa

J. EVANS  
C.C. ROUT  
D.A. ROCKE

#### References

1. ROBERTS RB, SHIRLEY MA. Reducing the risk of acid aspiration during caesarean section. *Anesthesia and Analgesia* 1974; 53: 859-68.
2. ROBERTS RB, SHIRLEY MA. Antacid therapy in obstetrics. *Anesthesiology* 1980; 53: 83.
3. TAYLOR G. Acid pulmonary aspiration syndrome after antacids. A case report. *British Journal of Anaesthesia* 1975; 47: 615-7.
4. RAIDOO DM, ROCKE DA, BROCK-UTNE JG, MARZALEK A, ENGELBRECHT HE. Critical volume for pulmonary acid aspiration: reappraisal in a primate model. *British Journal of Anaesthesia* 1990; 65: 248-50.
5. BOAG JW, HAYBITTLE JL, FOWLER JF, EMERY EW. The number of patients required in a clinical trial. *British Journal of Radiology* 1971; 44: 122-5.



### A problem with thiopentone solution

Thiopentone remains the anaesthetic induction agent of choice for Caesarean section under general anaesthesia.<sup>1</sup> Urgent anaesthesia may be required in obstetric practice and trays of labelled anaesthetic drugs are often kept in a refrigerator in the anaesthetic room.<sup>1</sup> I am reporting the solidification of thiopentone in a 20-ml syringe which was stored in such a refrigerator for 12 hours.

General anaesthesia was required for an emergency Caesarean section. The tray of anaesthetic drugs which had been prepared 12 hours previously was removed from the refrigerator. Induction of anaesthesia proceeded, but after 10 ml of 2.5% thiopentone had been given it became difficult to inject further solution despite a patent cannula and free running saline infusion. The dose injected induced anaesthesia and was followed by the rest of the anaesthetic sequence without further difficulty. When the syringe of thiopentone was examined soon after, the remainder of the syringe contents was observed to have solidified. The refrigerator used was specifically for drug storage and the

temperature gauge was registering in the safe range. The data sheet for thiopentone recommends that it is stable in solution for up to 24 hours after preparation. Studies have shown that a precipitate of thiopentone acid may appear in a 2.5% solution after 117 days at 5°C.<sup>2</sup> It is therefore an unexpected occurrence for the solution to solidify after only 12 hours, but it is recommended that drugs stored for emergency use in refrigerators should be inspected carefully before use.

Sunderland District General Hospital, M.A. THICKETT  
Sunderland SR4 7TP

### References

1. MOIR DD, THORBURN J. *Obstetric anaesthesia and analgesia* 3rd edn. London: Bailliere Tindall, 1986.
2. REYNOLDS JEF, ed. *Martindale. The extra pharmacopoeia*, 28th edn. London: The Pharmaceutical Press, 1982: 758-60.

### Epidural catheterisation

Epidural analgesia using a catheter technique is well established for pain relief and surgical anaesthesia. However, passage of the catheter beyond the tip of the Tuohy needle is frequently difficult and occasionally impossible. This may be due to direct obstruction to the passage of the catheter tip by duramater, and in such circumstances force exerted on the catheter may cause inadvertent dural puncture.

When this difficulty is encountered, we have found that pinching and bending the distal 3-4 mm of the catheter tip to an angle of 30 degrees is helpful in the passage of

catheter. All the holes for the exit of the drug lie proximal to this portion. The catheter is introduced with the angulation facing towards the bevel, as shown in the Figures. Using this technique, the failure rate has been greatly minimised.

Department of Anaesthesiology,  
Post Graduate Institute of  
Medical Education and Research,  
Chandigarh-160012,  
India

S.P. AMBESH  
A. KUMAR  
I. BHUKAL  
H. SINGH

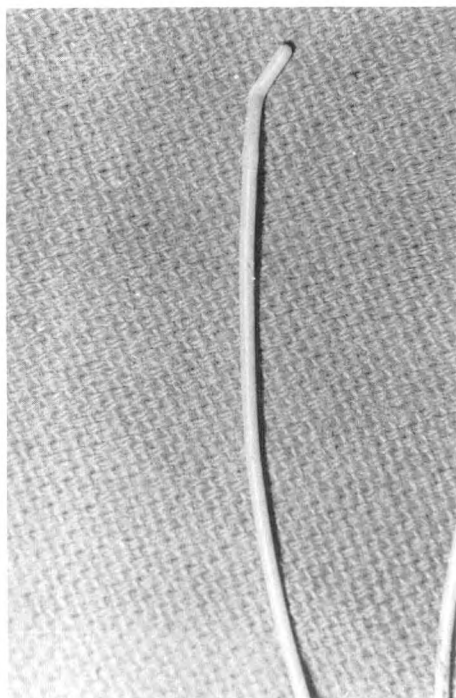


Fig. 1.

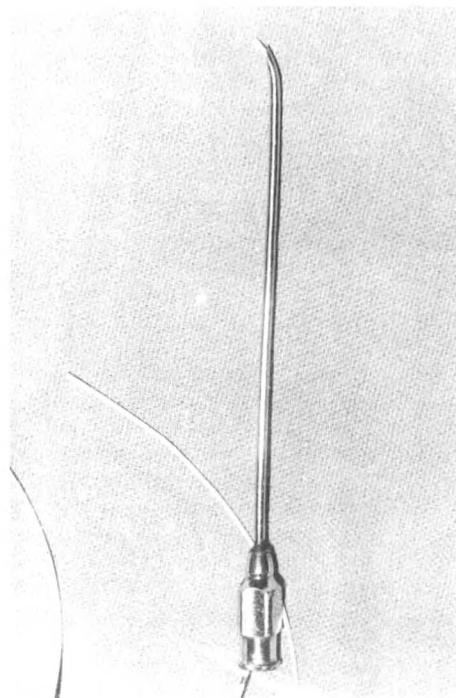


Fig. 2.

Neonatal up to 6.5kg **SIZE 1**

## Editor-in-Chief

Jan Eklund, Huddinge, Sweden

## Co-editors

Magna Andreen, Danderyd, Sweden  
Jørgen Viby-Mogensen, Copenhagen,  
Denmark

## Associate Editors

Barbro Ekström-Jodal, Gothenburg,  
Sweden  
Sven Erik Gisvold, Trondheim,  
Norway  
Bent Jubl, Aarhus, Denmark  
Matti Salo, Turku, Finland  
Thorsteinn Stefánsson, Reykjavik,  
Iceland

*Acta Anaesthesiologica Scandinavica* publishes papers on original work in anaesthesiology, intensive care, and subjects related to their basic sciences, conditional upon their being exclusive to this Journal. Case reports and short communications of particular interest will be considered for published material. Every effort will be made to expedite critical review of manuscripts to ensure rapid publication of papers of a high scientific standard.

## Subscription

One volume of eight issues annually.  
Subscription price 1991: DKK 880.00 including postage (GBP 82.00, DEM 244.00), USA, Canada and Japan: USD 173.00 including postage and air freight. All prices are payable in advance and subject to exchange-rate fluctuations.

In addition to the normal eight issues a number of supplements are delivered separately to subscribers free of charge.



**MUNKSGAARD  
INTERNATIONAL  
PUBLISHERS LTD**

COPENHAGEN • DENMARK

# Anaesthesiologica

*Iussu Societatis Anaesthesiologicae Scandinavicae Editi*

## ORDER FORM

Send to **Munksgaard International Publishers Ltd.**  
Postbox 2148, DK-1016 Copenhagen K, Denmark

OR **Munksgaard International Publishers Ltd.**  
c/o Publications Expediting Inc.  
200 Meacham Avenue, Elmont, NY 11003, USA

☐ I wish to subscribe to **Acta Anaesthesiologica Scandinavica** from 1991 and I enclose my personal cheque for \_\_\_\_\_

☐ Please bill me

☐ Please debit Eurocard / MasterCard / Access / VISA / American Express / Diners (delete as necessary)

Credit card no.

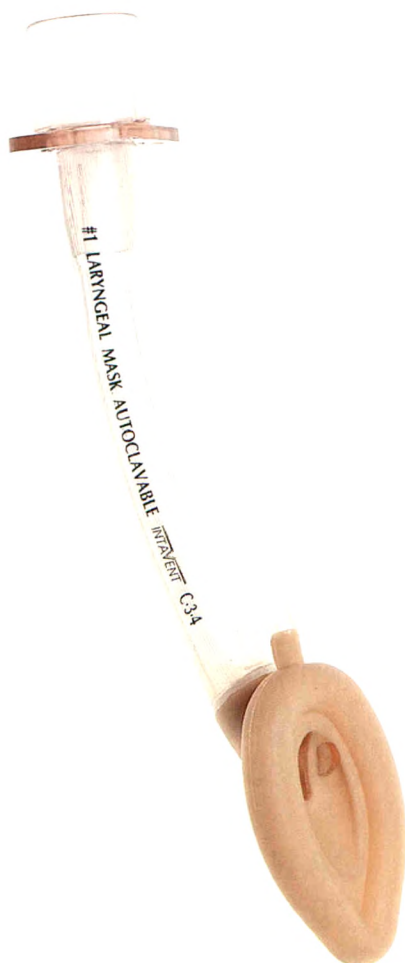
Expiry date \_\_\_\_\_ with the sum of \_\_\_\_\_

Signature \_\_\_\_\_ ☐ Please send me a free specimen copy

Name \_\_\_\_\_

Address \_\_\_\_\_

\_\_\_\_\_



SIZE 1



## Essential reading

from W.B.Saunders

### CLINICAL MONITORING

**Carol L. Lake**, *Prof. Anesthesiology, Univ. Virginia Medical School, Charlottesville, Virginia, USA.*

A superb review of all the latest devices and techniques used to monitor the anaesthetised patient's mechanical and physiological parameters during surgery.

You'll find comprehensive coverage of both invasive and noninvasive monitoring techniques, as well as automated and non-automated methods.

Thorough discussion of the specialised monitoring required in cardiac anaesthesia, neuroanaesthesia, obstetric anaesthesia, paediatric anaesthesia, intensive care, and more, brings you up to date in all aspects of monitoring practice.

- Includes detailed descriptions and colour figures of echocardiograms in various pathophysiological states.
- Describes monitoring of the anaesthetic delivering system
- Discusses the application of computers in patient acquisition
- Details pathophysiologic changes in measured variables expected in the perioperative period

#### CONTENTS

Electrocardiography: Monitoring for Ischaemia • Electrocardiography: Monitoring for Arrhythmias • Monitoring of Intravascular Pressures • Monitoring the Systemic Arterial Pressure • Monitoring Venous and Pulmonary Pressures • Intraoperative Echocardiography • Clinical Determination of Ventricular Function • Monitoring of Oxygen • Monitoring the Respiratory System • Intracranial Pressure • Electroencephalography • Evoked Potentials • Monitoring Peripheral Nervous System Function • Maternal and Fetal Monitoring • Computers in Monitoring • Patient Data Management Systems • The Anesthesia Machine • Monitoring Anesthetic Gases • Stethoscopy and Thermometry • Monitoring Hemostasis • Arterial Blood Gas Analysis • Biochemical Monitors.

**0 7216 2961 X 608pp 300 ills Hb 1989**  
**W.B. Saunders £67.85**

### ACUTE MANAGEMENT OF THE BURNED PATIENT

**J.A.J. Martyn**, *Dept Anesthesia, Massachusetts General Hospital, Boston, Mass., USA.*

A comprehensive new book covering all major aspects of anaesthesia and critical care of the burned patient, focusing on the stabilization of the patient and the establishment of a sterile, pain-free environment. Dealing with multidisciplinary skills, the chapters are derived from a multitude of sources ranging from transportation, through airway management and critical care to nutritional support and pain management.

- Includes coverage of new areas, such as management of toxic epidermal necrosis, aetiology of fluid management and treatment, and inhalation injury.
- Provides the best techniques for fluid, airway and pharmacological approaches to resuscitation.
- Covers psychiatric elements of burn trauma including specific aspects involving patients, relatives of patients and care givers.
- Outlines the rationale for excision and grafting
- Examines sensitivity to drugs in resuscitation, control of stress ulcers, control of infections and pain, and response/interaction to anaesthesia.

**0 7216 2774 9 368pp Hb May 1990**  
**W.B. Saunders £36.00**

### CARDIOPULMONARY CEREBRAL RESUSCITATION 3/e

**P. Safar & N.G. Bircher.**

Written by acknowledged international experts, this book belongs on the shelf of every physician and instructor in resuscitation.

**0 7216 2156 2 484pp 80ills Pb 1987**  
**W.B. Saunders (UK) £15.95**

**W.B. Saunders**  **Baillière Tindall**  
**HARCOURT BRACE JOVANOVICH**

Available from your bookseller. For more information please contact: The Marketing Dept, Harcourt Brace Jovanovich, 24-28 Oval Rd, London NW1 7DX, UK.  
OR phone: 081 300 3322

*Prices and specifications may vary without notice.*



### Subcutaneous morphine

We have noted with interest the recent correspondence concerning subcutaneous narcotics. Drs Jordan and Griffith's letter (*Anaesthesia* 1990; 45: 413) supports the traditional belief that the uptake of drugs from subcutaneous tissue is slow and erratic and states that 'the intramuscular route would be far superior'. Our recent study of the pharmacokinetics of subcutaneous morphine suggests that the uptake of morphine after subcutaneous injection is as rapid as after intramuscular injection.<sup>1</sup> We found peak blood morphine concentrations occurring at a mean of 17 (range 10–30) minutes after administration via an indwelling subcutaneous cannula. Comparative mean times to peak blood concentrations after intramuscular injection have been quoted to range from 17.5 to 27.8 minutes.<sup>2,3</sup> It thus appears that with reference to drug uptake there is little difference between administration of morphine either subcutaneously or intramuscularly.

One further positive aspect of the use of indwelling cannulae for administration of analgesic drugs applies equally well to both the subcutaneous or intramuscular route. This technique reduces the number of patient skin punctures to one, that occurring at cannula placement, and hence reduces the number of potentially contaminated

needles that need to be disposed of. In the current climate of heightened awareness of the risks of needlestick injuries to health workers, any means that can minimise these events should be encouraged. In our institution, the subcutaneous cannula has become the route of choice for postoperative analgesia for those patients in whom patient-controlled analgesia or epidural analgesia is not employed.

Royal Adelaide Hospital,  
Adelaide,  
South Australia 5000

T.J. SEMPLE  
P.E. MACINTYRE

### References

1. SEMPLE TJ, SOUTHALL EG, MACINTYRE PE, MATHER LE, RUNCIMAN WB. Pharmacokinetics of subcutaneous morphine in the elderly postoperative patient. *Pain* (Suppl.) 5: 5240.
2. KIRKPATRICK T, HENDERSON PD, NIMMO WS. Plasma morphine concentrations after intramuscular injection into the deltoid or gluteal muscles. *Anaesthesia* 1988; 43: 293–5.
3. RIGG JRA, BRAUNE RA, DAVIS C, KHADELWAL JK, GOLDSMITH CH. Variation in the disposition of morphine after IM administration in surgical patients. *British Journal of Anaesthesia* 1978; 50: 1125–30.

### The pump that gave too much: accidental overinfusion of prostacyclin

We report a case in which a 42-year-old female with Goodpasture's syndrome, having haemofiltration, accidentally received a bolus of epoprostenol.

Epoprostenol (formerly known as prostacyclin) is a naturally occurring prostaglandin produced by the intima of blood vessels and is the most potent inhibitor of platelet aggregation known. Its use is indicated as an alternative to heparin during renal dialysis, especially when a high risk of bleeding problems due to heparin exists. It is a potent vasodilator, the cardiovascular effects of which disappear within 30 minutes of the end of infusion.

The patient was being haemofiltered on an AK.10 haemodialysis system utilising a double-lumen catheter inserted into the subclavian vein for venovenous access. An epoprostenol infusion of 250 µg in 50 ml of sterile diluent was being infused from a Graseby syringe driver (M.S. 2000 type) at 2.5 ml/hour into the line taking the blood to the rotary pump. During the course of haemofiltration the system alarmed. The patient at this time became generally erythematous, profoundly hypotensive with the systolic arterial blood pressure decreasing from 120 to 60 mmHg and her central venous pressure decreasing from 10 to 6 mmHg. It was noted by the nursing staff at this time that the syringe of epoprostenol had suddenly emptied. Two units of hydroxyethyl starch, 10 ml of calcium gluconate 10%, and 15 mg of ephedrine were administered intra-

venously before her arterial blood pressure and central venous pressure were restored to their previous levels.

On investigation it was found that the flange on the barrel of the 50-ml syringe could, if the pressure in the system decreased below atmospheric pressure, be distorted sufficiently to disengage from the slot retaining the barrel in the Graseby pump, so that the barrel moved backwards towards the plunger thus emptying the syringe.

It is thought that a transient obstruction occurred to the lumen of the catheter in which the blood was being extracted, and since the rotary pump continued to function, the pressure at the point in the system at which the pump syringe was connected decreased sufficiently to empty the contents of the syringe into the circuit and thus into patient's circulation with the above results. Similar errors may occur whenever the barrel of a syringe in a syringe driver is inadequately restrained and the system is employed in a circuit in which it is possible to generate a subatmospheric pressure.

We understand that the manufacturers are in the process of modifying the Graseby pump to prevent such an error occurring in the future.

University Hospital of Wales,  
Cardiff CF4 4XW

J. DUNNE  
C. WISE

### Accidental intra-arterial injection of tubocurarine

A 28-year-old man presented for tonsillectomy. Despite premedication with temazepam 20 mg one hour previously, he remained very anxious in the anaesthetic room. Following a failed venepuncture due to venoconstriction, using a 22 gauge *In Syte* cannula a similar cannula was inserted in the left antecubital fossa. Tubocurarine 4 mg was given prior to the use of suxamethonium. Following the administration of 25 mg thiopentone 2.5%, the patient complained of intense pain in the left arm, which turned

pale. Intra-arterial injection was immediately suspected. His left radial pulse was present but was weaker than his right. The cannula was kept in place and hyaluronidase 1500 IU followed by 5 ml lignocaine 1% plain were injected intra-arterially before removal of the cannula. The patient now complained of weakness in his left lower arm and hand. Reduction in power to hand grip and adduction of the thumb and fingers was noted in comparison to the right hand.

The patient was reassured and anaesthesia was continued following insertion of a cannula into a right forearm vein; surgery proceeded without incident. Full power had returned to the left lower arm and hand 20 minutes following completion of surgery i.e. 45 minutes after the intra-arterial injection.

To date there has been no previous mention of intra-arterial injection of a nondepolarising muscle relaxant for prevention of suxamethonium pains. The patient in this case did not complain of any pain following injection of tubocurarine and it took about 4 minutes for him to notice any muscular weakness. Intra-arterial injection had already been suspected by this stage because of the pain felt following the thiopentone injection.<sup>1</sup> Perhaps more rapidly acting muscle relaxants such as vecuronium or atracurium should be used for pretreatment since any local muscle weakness, should it occur, would be observed more rapidly.<sup>2,3</sup> The patient should also be asked if there is any disturbance in muscle power following injection of a nondepolarising agent in these situations.

We would suggest that when using nondepolarising

muscle relaxants in similar situations, that in addition to asking about pain on injection, an enquiry with regard to muscle weakness be included and further suggest the use of vecuronium or atracurium for pretreatment since any weakness will be noticed more rapidly.

Waveney Hospital,  
Ballymena,  
Co. Antrim BT43 6HH

E. DEVLIN  
I. BALI

#### References

1. WATERS DJ. Intra-arterial Thiopentone: a physico-chemical phenomenon. *Anaesthesia* 1966; **21**: 346–56.
2. MANCHIKANTI L, GROW JB, COLLIVER JA, CANELLA MG, HADLEY CH. Atracurium pretreatment for succinylcholine-induced fasciculations and postoperative myalgia. *Anesthesia and Analgesia* 1985; **64**: 1010–14.
3. FERRES CJ, MIRAKHUR RK, CRAIG HJL, BROWNE ES, CLARKE RSJ. Pretreatment with vecuronium as a prophylactic against post-suxamethonium muscle pain. *British Journal of Anaesthesia* 1983; **55**: 735–40.

#### Persistent erection and general anaesthesia

Your correspondent, Dr Hutchison (*Anaesthesia* 1990; **45**: 794) rightly recognises that the problem of persistent erection under general anaesthesia is a rare occurrence. However, we have noticed an increasing incidence in our practice of neuroanaesthesia corresponding with a change in technique. Over the past 2 years, we have been using a dose step-down technique of intravenous anaesthesia (similar to that described by Prys-Roberts) for craniotomy. After induction, tracheal intubation and arterial cannulation, it is our custom to catheterise these patients. We have noticed that under the stimulation of catheterisation, male patients frequently have an erection which can complicate the procedure.

Emergence from propofol anaesthesia has been associated with hallucinations and amorous advances from female patients.<sup>1,2</sup> It has been suggested that the hallucinations may be related to alterations in cerebral neurochemistry. The mechanism of penile erection under general anaesthesia is complex, but is attributed to a combination of psychogenic and reflexogenic stimuli. An incidence of 1% was reported in an American paper using high-dose

fentanyl (40–50 µg/kg) infusion,<sup>3</sup> but we would estimate our incidence to be higher. It would seem that propofol may have excitatory effects in arousal centres in the brain which could explain both phenomena of increased incidence of penile erection in men and arousal in women.

We would be interested to learn if any other workers have noticed this problem and particularly if the continuous infusion technique is proving useful in urology.

Southern General Hospital,  
Glasgow G51 4TF

H.E. HOSIE  
J.G. TODD

#### References

1. LLOYD EL. Hallucinations under anaesthesia. *Anaesthesia* 1987; **42**: 1015–6.
2. HUNTER DN, THORNILY A, WHITBURN R. Arousal from propofol. *Anaesthesia* 1987; **42**: 1128–9.
3. ROY R. Cardiovascular effects of ketamine given to relieve penile tumescence after high doses of fentanyl. *Anesthesiology* 1984; **61**: 610–13.

#### Hazards from homely anaesthetic rooms

It has become popular to attempt to allay patients' fears as much as possible. This has included decorating anaesthetic rooms in ways which are believed to be less threatening than usual. There are dangers in taking this to excess. I should like to record a cautionary tale of one such instance. While preparing for an operating list on a Wednesday afternoon the alert senior house officer working with me commented that the suxamethonium felt very warm. I immediately looked for the refrigerator temperature gauge, initially without success. Then I realised that a large, plastic 3 D 'Minnie Mouse' which had been stuck on the door was obscuring the gauge. When this had been removed the gauge was shown to be recording a very high temperature

because the switch located behind a test-tube rack had been turned off. Enquiries revealed that this situation had existed at least since the previous weekend. It was considered necessary to throw away the entire contents of the refrigerator which included a large stock of Helonid as well as suxamethonium and atracurium.

Fortunately no patient was harmed, but this event was a reminder that all gauges serve an essential purpose and must under no circumstances be obscured, even in the case of making an anaesthetic room more inviting.

Leicester Royal Infirmary,  
Leicester LE1 5WW

R.H. JAMES



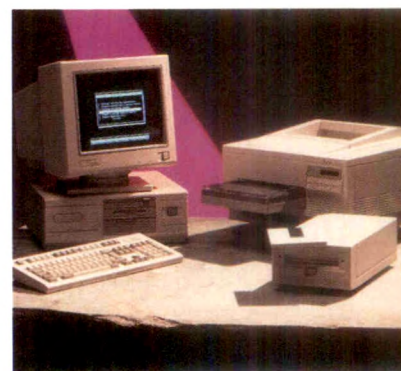
# The ARC system: taking care of the data while you take care of the patient.

The ARC system offers the complete data management facility for the anaesthetist in theatre.

With any other computerised management system the two most time consuming aspects are data entry and data output.

The ARC system deals with both of these problems at source, allowing the effortless combination of monitored data and procedural information.

Automatic data logging, SMAI keys and full networking minimise data input. Leaving you more time to deal with your patient.

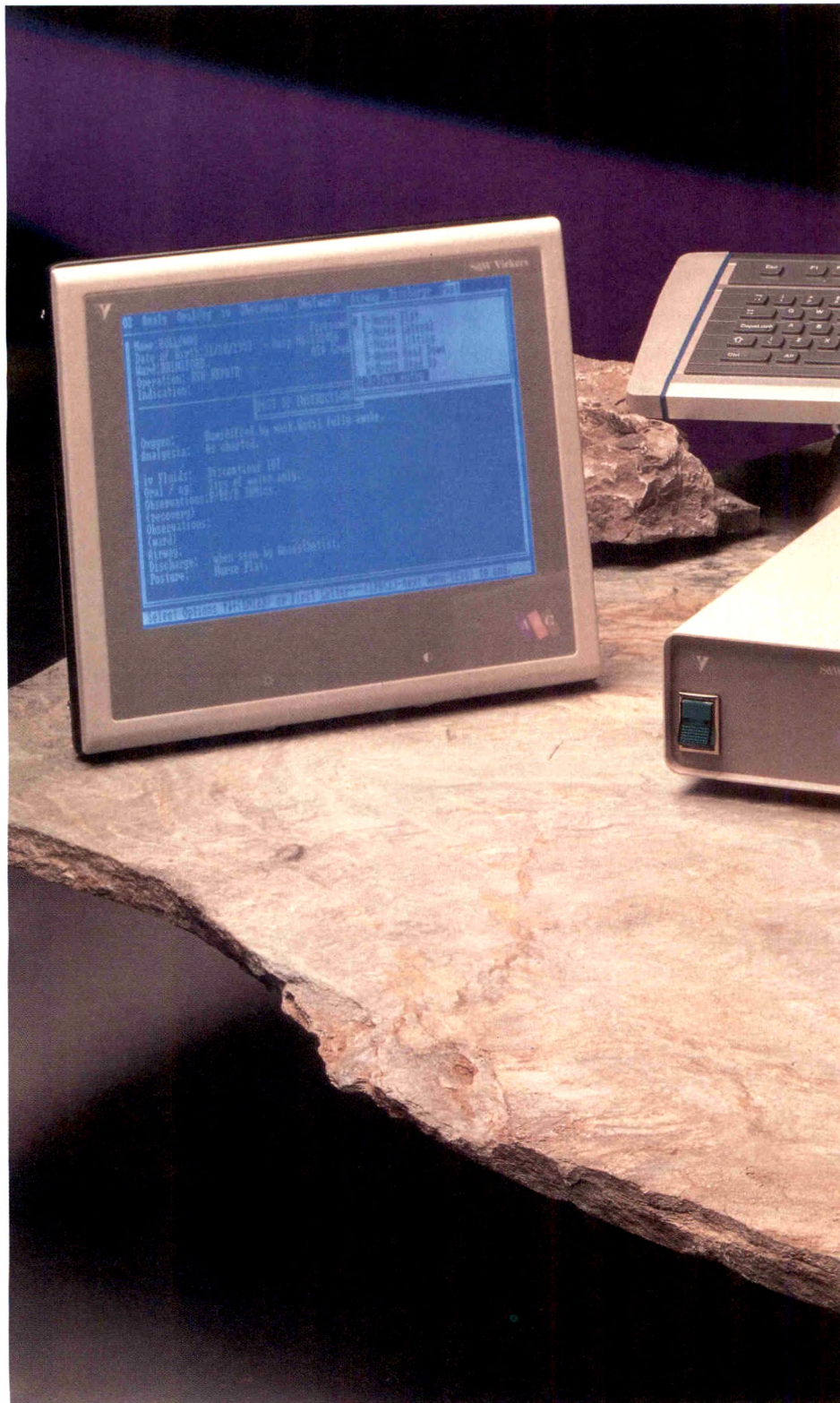


Later, when the pressure's off you can print out patient records at ARC-central.

Additionally, theatre usage, Körner, morbidity and *ad hoc* reports can all be generated with laser quality output whenever you need them.

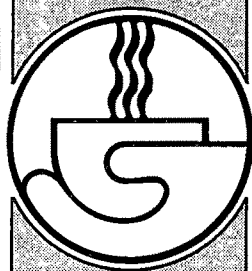
The ARC system — you can't always choose the data you manage but you can choose how you manage it.

For further details on the ARC system contact S&W Vickers at any of the addresses below.



## S&W Vickers

Ruxley Corner, Sidcup, Kent, DA14 5BL. Tel 081-309 0433 Fax 081-309 0919  
32 Kerse Road, Stirling, FK7 7TA. Tel 0786 51602 Fax 0786 51603  
4 Belmont Business Park, Ballymore, Belfast. Tel 0232 660766 Fax 0232 660772



**Editor:**  
John G. Roberts,  
Adelaide

**Associate Editor:**  
Jeanette Thirlwell,  
Sydney

**Assistant Editor-  
Intensive Care:**  
A.W. Duncan,  
Perth

**Editorial Committee:**  
Barry Baker,  
Dunedin, NZ

Kester Brown,  
Melbourne

Noel M Cass,  
Melbourne

John N. Ditton,  
Sydney

Anthony Ilsley,  
Adelaide

John H Overton,  
Sydney

Teik Oh,  
Hong Kong

John D Paull,  
Melbourne

Phelim Reilly,  
Brisbane

Walter R Thompson,  
Perth

# ANAESTHESIA AND INTENSIVE CARE

*Journal of the Australian Society  
of Anaesthetists*

Anaesthesia and Intensive Care is a quarterly journal (published Feb 1, May 1, Aug 1 and Nov 1) presenting articles of scientific and clinical interest in the specialities of Anaesthesia, Intensive Care Medicine and related disciplines. Frequent symposium issues, comprehensive review articles, dissertations on the history of anaesthesia and education and training in the specialities are very informative, while our correspondence section gives contributors an opportunity to express point of view. Future meetings, both Australian and International, are listed.

The Journal is international in content and readership and offers very reasonable subscription rates with American Express and Visa facilities.

Advertising Rates available on request.

## ANNUAL SUBSCRIPTION RATES (1991 rates)

- |                                    |                             |                      |
|------------------------------------|-----------------------------|----------------------|
| ★ Within Australia – surface mail  | :Aust \$56.00               | back issues: \$14.00 |
| ★ Outside Australia – surface mail | :Aust \$84.00               | back issues: \$21.00 |
| – air mail                         | :Rates available on request |                      |

Send orders to: ANAESTHESIA AND INTENSIVE CARE  
P.O. Box 600 Edgecliff, NSW 2027, AUSTRALIA Phone (02) 327 4022 Fax (02) 327 7666

*I wish to subscribe to ANAESTHESIA AND INTENSIVE CARE ISSN: 0310 - 057X commencing  
with the current volume*

NAME .....  
(BLOCK LETTERS PLEASE)

ADDRESS .....

.....POSTCODE .....

COUNTRY.....DATE .....

☐ (✓) PLEASE SEND BY SURFACE MAIL

☐ (✓) MY PAYMENT IS ENCLOSED

☐ (✓) PLEASE SEND ME INFORMATION  
ON AIR MAIL RATES FOR MY REGION

☐ (✓) PLEASE INVOICE ME WITH THE  
FIRST ISSUE

### FOR PAYMENT BY CREDIT CARD

Please charge my

☐ Visa ☐ American Express

Cardholder's Name .....

Credit Card No.....

Signature.....Expiry Date.....

**Fenem CO<sub>2</sub> detector device**

The Fenem CO<sub>2</sub> detector described by Denman *et al.* (*Anaesthesia* 1990; **45**: 465-7) is a worthwhile aid in our defence against oesophageal intubation and litigation. It is cheap, simple, reliable and almost failsafe. Unfortunately, like the capnograph, the oesophageal detector, the fibreoptic bronchoscope and the Eschmann tracheal tube introducer, it is fundamentally flawed since it is not permanently fixed to the tracheal tube and is therefore liable to be absent when most needed. However, it does point the way towards a nearer ideal: an inexpensive,

tracheal tube impregnated with a nontoxic chemical that instantly changes colour when exposed to more than 1% carbon dioxide. Let us hope the designers and manufacturers of medical equipment will be able to meet this challenge.

*Royal Perth Hospital,  
Perth,  
Western Australia.*

J.R. BRIMACOMBE



## Book reviews

- Manual of cardiorespiratory critical care** 78  
F. GUZMAN, M.A. HEDLEY BROWN, M. BEEN, S. COOK, C. WREN AND D. RICHENS  
**Clinical respiratory physiology** 78  
A.E. TAYLOR, K. REHDER, R.E. HYATT AND J.C. PARKER

- Fiberoptic airway endoscopy in anaesthesia and critical care** 79  
Edited by A. OVASSAPIAN

### Manual of cardiorespiratory critical care

F. GUZMAN, M.A. HEDLEY BROWN, M. BEEN, S. COOK, C. WREN, AND D. RICHENS. Pp. xi+314. Butterworth Scientific, 1989. £19.50.

This multi-author paperback is intended for junior medical and nursing staff, although for reference, not uninterrupted study. It is very comprehensive with numerous line drawings, and its eight sections cover cardiac medicine, cardiac surgery in all its aspects, pulmonary medicine, thoracic surgery and paediatric cardiac and pulmonary medicine and surgery. The preface states 'the emphasis is on practical instant help in acute situations', so I looked up a few topics on which the inexperienced might welcome immediate guidance, namely cardiac arrest, cardiac tamponade, a low cardiac output state after cardiac surgery, initiating mechanical ventilation, brain damage and septic shock. The section on cardiac arrest includes the classic, but increasingly outdated ABC sequence with the usual diagrams, hardly appropriate for the intensive care situation, whilst what little information on drug therapy is given is largely obsolete. The differential diagnosis and suggestive signs of cardiac tamponade, a very difficult problem, is barely touched on.

There is much about respiratory function, but the criteria for deciding when to initiate mechanical ventilation are far too sketchy to help the tyro, and arbitrary guidelines would be much more helpful. Incidentally, the continuing 30-year-old bedside confusion between respiratory function and respiratory fatigue and the practical problems of intensive care of acute respiratory failure are well illustrated here, where there is an insistence that 'respiratory failure is defined in terms of function, *not* in terms of respiratory mechanics . . .', whilst on the other hand (and in my opinion correctly) 'fatigue . . . is often the critical factor in precipitating (mechanical) intervention'. The discussion on postoperative low cardiac output state is not helpful for again, although there is a list of signs (far from comprehensive), there is little or nothing about its management. So far as brain damage is concerned, there is no evidence that high dosage steroids are helpful, even if there is cerebral oedema (if the patient recovers this is unlikely to occur) and good evidence from head trauma shows they are actively harmful. Septic shock is the best of this disappointing bunch, but again treatment is barely described and there is no mention of inotropes, in particular noradrenaline and dobutamine which have been widely advocated in the literature in the past few years. Noradrenaline by the way, for those not closely involved in resuscitation and intensive care, has again become a 'respectable', widely used drug, despite Ahlquist's invaluable but baleful classification of sympathomimetic agents, 40 years ago. On the other hand, some sections, including postoperative infection and paediatric postoperative care (I am not qualified to speak on

neonatal management), are most helpful. The index is adequate and there is a short list of references, although none is from the very extensive literature on anaesthesia or intensive care on the subject. By today's standards this book is not expensive.

Despite a critic's carping, it contains much useful, often tabulated, information and anyone who intends taking a serious interest in this field would find it a helpful addition to his/her bookshelf, dipping into it here and there in the twilight hours, although they might like to wait for the next, improved edition. For the moment a beta at most.

A. GILSTON

### Clinical respiratory physiology

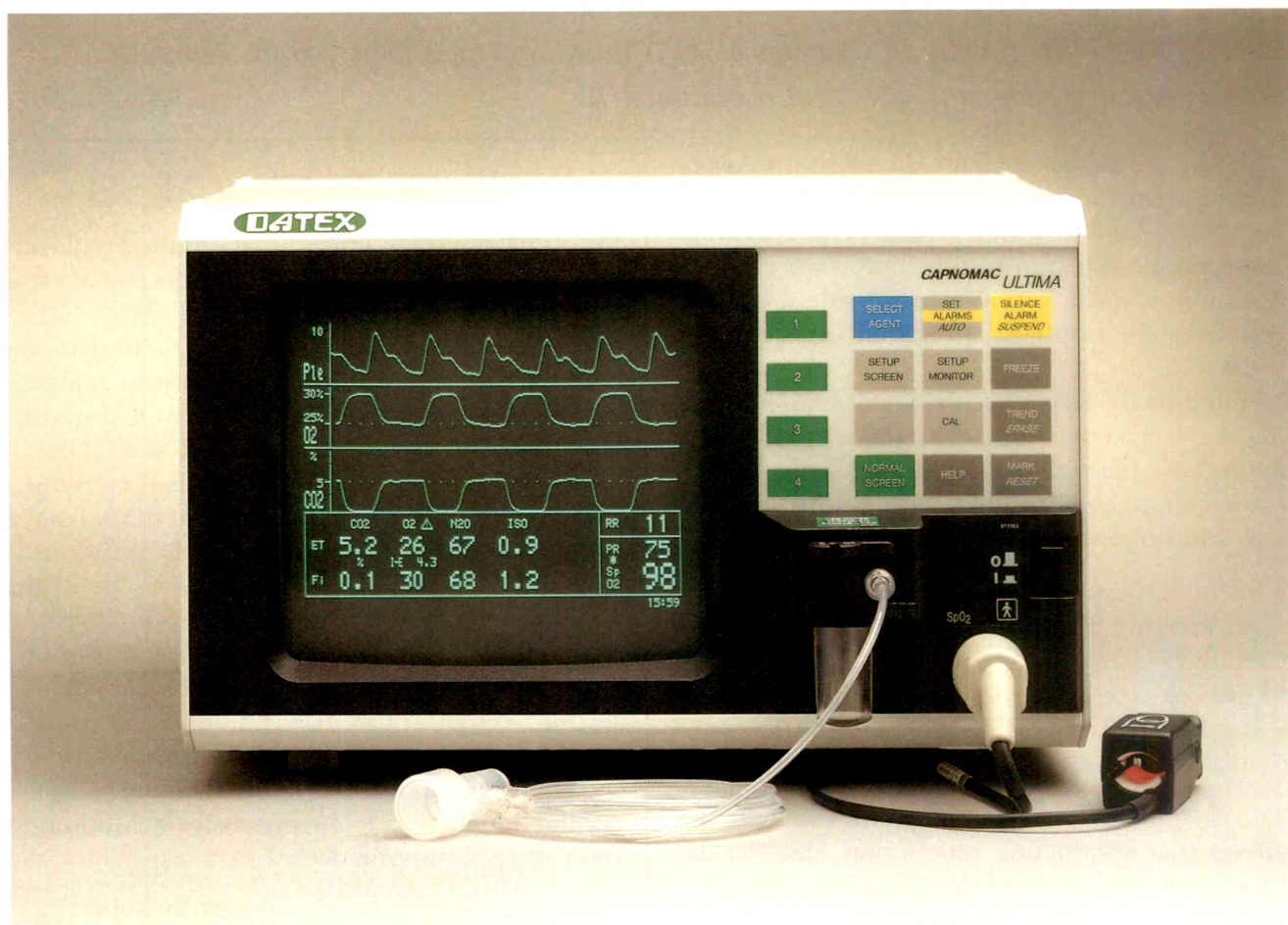
A.E. TAYLOR, K. REHDER, R.E. HYATT AND J.C. PARKER. Pp. x+300. W.B. Saunders, 1990. £14.50.

This paperback sets out to describe the basic principles of respiratory physiology and their underlying mechanisms to medical undergraduate and postgraduate students. Many of these readers will have used West's *Respiratory physiology—the essentials* but the two texts, although similar at first glance, are quite different. This book is packed with more information, more up-to-date in places, but considerably less digestible.

It covers all conventional aspects of the subject and achieves an admirable cohesion between the four authors. Nonetheless, some areas are stronger than others. The first chapter is an overview of lung structure and function that is marvellously readable. Chapters on the mechanics of breathing, pulmonary and bronchial circulations and pulmonary fluid exchange are particularly good, the latter being of special interest to anaesthetists and not easy to find elsewhere in concise form. Assessment of lung function is well covered, and the book ends with a useful chapter on altitude and diving physiology.

The rest of the book is less easy to commend. The subject is quantitative and thus condemned to be understood best by those with a feel for graphical or algebraic analysis. The diagrams are adequate, with some useful electron-micrographs, but they do not make up for a text that is frequently heavy. In some places (e.g. CO<sub>2</sub> transport) the discussion actually proceeds in reverse order of importance. The important chapter on alveolar ventilation-perfusion is perhaps the most difficult of all. It is asking too much of any reader to have to plough through an explanation of gas and blood R lines before discovering that regional ventilation-perfusion ratio inequalities even exist. The alveolar gas equation is introduced baldly with little explanation of its importance, intuitive derivation or application to gases other than oxygen. Some areas of

# The Datex Ultima - when you need the ultimate in safe anaesthesia care.



The new Datex Ultima is the flexible response to tightening monitoring standards.

It integrates all the parameters of gas exchange *and* ventilator function helping you to ensure complete patient safety during anaesthesia.

The new Ultima measures and displays CO<sub>2</sub>, O<sub>2</sub>, N<sub>2</sub>O and anaesthetic agents on the large, easy-to-read screen.

And since patient safety in routine anaesthesia is primarily dependant on adequate oxygenation, the fast paramagnetic Oxygen sensor in the Ultima measures inspired *and* expired O<sub>2</sub> data together with a breath-by-breath waveform display.

Additionally, with the new Ultima you can add pulse oximetry, agent identification, airway pressure and tidal volume – as and when you need them.

The Ultima also incorporates the innovative D-fend membrane water-trap eliminating the moisture problems which plague other airway gas monitors.

To find out how the new Datex Ultima can help you provide safe anaesthesia care ring your local S&W Vickers office on the number below.

## S&W Vickers

Ruxley Corner, Sidcup, Kent, DA14 5BL. Tel 081-309 0433 Fax 081-309 0919

32 Kerse Road, Stirling, FK7 7TA. Tel 0786 51602 Fax 0786 51603

4 Balmoral Business Park, Boucher Crescent, Belfast. Tel 0232 669266





# EPIDURAL & SPINAL BLOCKADE IN OBSTETRICS



Edited by

**Felicity Reynolds**, *Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.*

This book has evolved from the Obstetric Anaesthetists' Association meeting in the UK in September 1989. Unlike other books of proceedings however, the well known contributors were invited not only to speak but also to provide a chapter reviewing their subject, prepared especially for the book.

Edited by Dr. Reynolds, the result is an interesting blend of a review of the last ten year's developments coupled with presentations of current research and coloured throughout with stimulating, amusing discussion.

- Includes unique illustrations, using state-of-the-art imaging techniques, of the anatomy of the epidural space.
- Features excellent reviews of the rapidly expanding fields of Caesarean section and the use of spinal opioids.

The closing section, The Recipient, Discusses the effects that conduction blocks may have on the baby, and reviews the lay literature covering consumers views of epidural practice.

## C O N T E N T S

INTRODUCTION: The lumbar epidural region: anatomy and approach • Indications and contraindications for epidural blockade. MANAGEMENT OF LABOUR: Test doses • Epidural analgesia by continuous infusion • Epidural analgesia and the management of the second stage of labour: a failure to progress. COMPLICATIONS: Local anaesthetic toxicity • Lumbar epidural analgesia in obstetrics: taps and patches • Total spinal anaesthesia: the effect of spinal infusions. CAESAREAN SECTION: Regional or general anaesthesia for caesarean section • Spinal and/or epidural blockade for Caesarean section • Factors modifying epidural block for Caesarean section. SPINAL OPIOIDS: Epidural and spinal opiates in labour • Spinal opioids for Caesarean section. THE RECIPIENT: Effects on the baby of conduction blockade in obstetrics • Mothers' views of epidural practices.

0 7020 1401 X 238pp Ills Pb September 1990 £17.50

**Baillière Tindall**

### ORDERING INSTRUCTIONS

Available through booksellers. In case of difficulty, complete the order form and send, with payment, to:

**Marketing Department, Harcourt Brace Jovanovich, 24-28 Oval Road, London NW1 7DX, UK.**

Prices, specifications and availability are subject to change without notice. Postage/Packing/Handling Charges - UK & Ireland: Prices quoted include postage/packing/handling charges for delivery within the UK & Ireland. Europe: Add £2.50 for the first book and £0.75 for each additional book.

Rest of World: Add £5.00 for the first book and £1.50 for each additional book.

Delivery overseas 28 days minimum (air-speeded methods used).

OR phone your order to customer services on (081) 300 3322

Please send \_\_\_\_\_ copy/ies of Reynolds, Epidural and Spinal Blockade in Obstetrics.

I enclose £\_\_\_\_\_ in payment for books and  
£\_\_\_\_\_ for postage/packing (see note above)

I enclose a total of £\_\_\_\_\_ being payment by  
(please tick):

☐ Cheque (made payable to *Harcourt Brace Jovanovich*)

☐ American Express ☐ Diners Club

☐ Eurocard/Access/Mastercard

☐ Visa/Barclaycard/Bankamericard

Card Number \_\_\_\_\_

Expiry Date \_\_\_\_\_

Daytime phone no. \_\_\_\_\_

Name (please print) \_\_\_\_\_

Address \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

Signature \_\_\_\_\_ Date \_\_\_\_\_

current interest, such as the assessment of respiratory muscle function, are not covered.

Each chapter has suggestions for further reading although over half of these references are earlier than 1980, and some even refer to the *Handbook of physiology* published in the 1960s and now superseded. There are also questions and answers based on each chapter's material, and an adequate index. It is a book one might buy with mild foreboding rather than excitement, as the ratio of work put in by the reader to the understanding obtained would be uncomfortably high. I would stick to West, and dip into the pleasures of Nunn's *Applied Respiratory Physiology* for further exploration.

N.J.H. DAVIES

#### **Fiberoptic airway endoscopy in anesthesia and critical care**

Edited by A. OVASSAPIAN. Pp. xix + 172. Raven Press, 1990. \$126.00.

Professor Ovassapian has done us a great service by this timely and excellent basic textbook on fiberoptic endoscopy for anaesthetists. There is a pressing need for a basic textbook in this field for the novice and the teacher and this fills that need most appropriately. At 172 pages and 11 chapters it is a slim volume so that the reader can go to those chapters of particular interest. The photographs through endoscopes are outstanding and the illustrations

demonstrating anatomy and explaining techniques make the book a great resource.

The opening three chapters are on the basic principles of fiberoptic technology, the anatomy of the airway, and the radiology of the airway. These aspects are often overlooked by the novice in the keenness to acquire clinical expertise. A whole chapter on topical anaesthesia indicates the importance that USA anaesthetists give to awake tracheal intubation. Perhaps we on this side of the Atlantic undervalue the advantages inherent in awake fiberoptic tracheal intubation. Such differences in philosophy in no way diminish the value of this book for anaesthetists in Great Britain and Ireland. Each chapter is followed by an extensive list of references; for example, the chapter on fiberoptic tracheal intubation has 64—a most valuable resource for those wishing to read more widely.

The chapters on the use of the fibrescope in thoracic anaesthesia for bronchial intubation, and its application in the intensive care unit, will be of particular interest to a specialist group of anaesthetists. The chapters on the difficult airway and the difficult intubation put the fibrescope into context. A clinical approach is presented with a number of options available of which the fibrescope is one. Professor Ovassapian's wide experience as a teacher is reflected in his valuable chapter on teaching and learning fiberoptic tracheal intubation. A chapter devoted to 10 case reports makes most interesting reading and demonstrates that this book is written from a wealth of clinical experience. I am sure that this will be a book to be recommended for several years to come.

R.M. TOWEY



## Obituaries

**Butt-Kwan, N.S.G., FCAaes.** Latterly a Consultant Anaesthetist in Hong Kong. Qualified from the University of Otago in 1947.

**Knowles, G.S.A., MRCS, LRCP, MB, BS, FCAaes.** Formerly Senior Consultant Anaesthetist at the North Middlesex Hospital. Qualified from the University of London in 1938.

**Young, H.S.A, MB, BCh, BAO, FFARCSI.** Formerly Consultant Anaesthetist at the Royal Victoria Hospital. Qualified from Queen's University, Belfast in 1968.

## International congress calendar

### 1991

**10-13 January.** Miami. *28th Annual Post-Graduate Seminar in Anesthesiology.*

*Information:* Barbara McNulty, Continuing Education Programs, 7480 Fairway Drive, Suite 106, Miami Lakes, Florida 33014, USA.

**12-19 January.** Barbados. *9th Annual Symposium: clinical update in Anesthesiology.*

*Information:* Ms H. Phillips, Mount Sinai Medical Center, 1 Gustave L. Levy Place, Box 1010, New York, NY 10029, USA.

**15-17 January.** Doha-Qatar. *First Gulf Conference on Intensive Care Medicine.*

*Information:* Dr Jamal S. Al-Shanableh, Consultant Cardiac Anaesthesiologist, Secretary, P.O. Box 3050, Hamad General Hospital, Doha-Qatar.

**17-19 January.** London. *Winter Scientific Meeting and Technical Exhibition Queen Elizabeth Conference Centre.*

*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

**25-27 January.** Los Angeles. *Twenty-ninth Clinical Conference in Pediatric Anesthesiology.*

*Information:* Katherine Barky, MD, Program Director, Division of Anesthesiology, Children's Hospital of Los Angeles, P.O. Box 54700, Los Angeles, California 90054, USA.

**26 January-1 February.** Colorado. *Update on Anesthesia.*

*Information:* C.M. Ballinger, MD, Office of Continuing Medical Education, Campus Box C-295, 4200 East Ninth Avenue, Denver, Colorado 80262, USA.

**2-9 February.** Colorado. *17th Annual Vail Conference in Anesthesiology.*

*Information:* Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101, USA.

**9-11 February.** Colorado. *Anesthesia Update-1991.*

*Information:* Alan H. Goldberg, MD, PhD, Department of Anesthesiology, Medical College of Wisconsin, 8700 West Wisconsin Avenue, Milwaukee, Wisconsin 53226, USA.

**9-16 February.** Vail. *16th Annual Symposium in Intensive Care.*

*Information:* Professional Seminars, P.O. Box 012318, Miami, FL 33101, USA.

**9-16 February.** Colorado. *Aspen Anesthesia-1991.*

*Information:* Holiday Seminars, 755 Long Hollow Pike, Goodlettsville, Tennessee 37072, USA.

**15-17 February.** India. *Critical Care Workshop.*

*Information:* Dr Sushila Shah, Department of Anaesthesiology Tata Memorial Hospital, Bombay-400012, India.

**22-24 February.** Texas. *Refresher Course in Anesthesiology.*

*Information:* Eason Cockings, MD, Department of Anesthesiology, Texas Tech University Health Sciences Center, 3601 Fourth Street, Lubbock, Texas 79430, USA.

**8-12 March.** San Antonio. *65th Congress of the International Anesthesia Research Society.*

*Information:* Emerson A. Moffitt, IARS, 3645 Warrensville Center Road, Cleveland, Ohio 44122, USA.

**9-14 March.** Pretoria. *The 1991 National Anaesthetic Congress of the South African Society of Anaesthetists.*

*Information:* Professor J.M. Hugo, Chairman, Department of Anaesthetics, Faculty of Medicine, P.O. Box 667, Pretoria, South Africa.

**17-18 March.** Brussels. *Workstation Concept for Intensive Care and Anaesthesia. Satellite meeting to the Eleventh International Symposium on Intensive Care and Emergency Medicine.*

*Information:* Mrs Smits-de Smet, Department of Intensive Care, Erasme Hospital, Route de Lennik, 808 B-1070 Brussels, Belgium.

**19-22 March.** Brussels. *11th International Symposium on Intensive Care and Emergency Medicine.*

*Information:* Professor J.L. Vincent, Department of Intensive Care, Erasme University Hospital, Route de Lennik 808, B-1070 Brussels.

**28-30 March.** Osaka. *38th Annual Meeting of the Japan Society of Anesthesiology.*

*Information:* Professor M. Fujita, Japan Society of Anesthesiology, TY Building, 18-11 Hongo 3-chome, Bunkyo-Ku, Tokyo 113, Japan.

**4-7 April.** Cincinnati. *16th Annual Meeting of the American Society of Regional Anesthesia.*

*Information:* P.O. Box 11086, Richmond, Virginia 23230-1086, USA.

**3-5 April.** Oxford. *Junior Anaesthetists' Group of the Association of Anaesthetists of Great Britain and Ireland Linkman Conference and Annual Scientific Meeting.*

*Information:* Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

**11-13 April.** Belgium. *Update in Cardiac Surgery, Anaesthesia and Intensive Care.*

*Information:* The Secretary, Department of Anesthesia B11-5 de Pintelaan 185, B9000 Gent, Belgium.

**15-18 April.** Hamamatsu, Japan. *6th International Symposium on Computing in Anesthesia and Intensive Care.*

*Information:* Dr K. Ikeda, Chairman of the Organising Committee, c/o Department of Anesthesiology, Hamamatsu University School of Medicine, 3600 Handa-cho, Hamamatsu, Shizuoka, 431-31 Japan.

**17-21 April.** Antilles. *19th International Society on Oxygen Transport to Tissue.*

*Information:* Professor W. Erdmann, Department of Anaesthesiology, Dr Molewaterplein 40, 3015 GD Rotterdam, The Netherlands.

**18-20 April.** Paris. *European Academy Open Scientific Meeting.*

*Information:* Professor J.C. Otteni, Service d'anesthésie et de réanimation, Hôpital de Hautepierre, Avenue Molière, F-67098 Strasbourg, France.

**18-21 April.** Paris. *European Academy of Anaesthesiology. Refresher Course.*

*Information:* Professor J.M. Desmonts, Département d'Anesthésie, Hôpital Bichat, 46 rue Henri-Huchard, 75018 Paris, France.

**23-27 April.** Montreal. *Second International Symposium on Pediatric Pain.*

*Information:* Pain Secretariat, 3450 University Street, Montreal, Quebec, H3A 2A7, Canada.

**3-5 May.** Philadelphia. *AUA Annual Meeting.*

*Information:* Stephen J. Prevotnik, Department of Anesthesia,

- Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104, USA.
- 4-8 May.** San Antonio. *Society of Cardiovascular Anesthetists*.  
Information: P.O. Box 11086, Richmond, Virginia 23230-1086, USA.
- 5-12 May.** Sydney. *Faculty of Anaesthetists, R.A.C.S., General Scientific Meeting*.  
Information: Faculty of Anaesthetists, Royal Australasian College of Surgeons, Spring Street, Melbourne 3000, Australia.
- 9-12 May.** Washington DC. *6th International Dental Congress on Modern Pain Control*.  
Information: American Dental Society of Anesthesiology, Inc., 211 E. Chicago Avenue, Suite 948, Chicago, IL 60611, USA.
- 16-19 May.** California. *Californian Society of Anesthesiologists Annual Meeting*.  
Information: Californian Society of Anesthesiologists, 1065 East Hillsdale Boulevard, Suite 410, Foster City, California 94494, USA.
- 18-19 May.** Taiwan. *1st Asian-Oceanic Symposium on Regional Anaesthesia*.  
Information: Professor J.H. Lee, P.O. Box 26-473 Taipei, Taiwan 10713.
- 24-25 May.** The Netherlands. *Receptors of the Brain, Lung and Heart: State of the Art*.  
Information: Cader Research B.V., P.O. Box 85, 4854 ZH Breda/Bavel, The Netherlands.
- 27-31 May.** Montreal. *McGill University Annual Review Course in Anaesthesia*.  
Information: Post Graduate Board, Royal Victoria Hospital, 687 Pine Avenue West, Room H308, Montreal Quebec, H3A 1A1, Canada.
- 1-2 June.** Texas. *Eighth Annual Pain Symposium*.  
Information: James E. Heavner, DVM, PhD, Department of Anesthesiology, Texas Tech University Health Sciences Center, 3601 Fourth Street, Lubbock, Texas 79430, USA.
- 4-7 June.** Milano. *6th Annual Meeting of the European Association of Cardiothoracic Anaesthesiologists*.  
Information: Francesca Rovelli, The Organizing Secretariat O.I.C. Incentive, Viale Majno, 21, 20122 Milano, Italy.
- 13-16 June.** Florida. *Florida Society of Anesthesiologists Annual Meeting*.  
Information: Florida Society of Anesthesiologists, 3000 34th Street South, Suite F, St. Petersburg, Florida 33711, USA.
- 21-25 June.** Quebec City. *48th Annual Meeting of Canadian Anaesthetists' Society*.  
Information: Ms Ann Andrews, CAS, 187 Gerrard St. E., Toronto, Ontario M5A 2E5, Canada.
- 24-28 June.** Norway. *21st Congress of the Scandinavian Society of Anaesthesiologists*.  
Information: Department of Continuing Education, The Norwegian Institute of Technology, N-7034 Trondheim, Norway.
- 22-24 August.** Auckland. *The Annual Conference of New Zealand Anaesthetists*.  
Information: Department of Anaesthesia, Auckland Hospital, Park Road, Auckland, New Zealand.
- 28 August-1 September.** Strasbourg. *European Academy Scientific Meeting*.  
Information: Professor J.C. Otteni, Service d'anesthésie et de Réanimation, Hôpital de Hautepierre, Avenue Molière, F-67098 Strasbourg, France.
- 4-8 September.** Rio de Janeiro. *XXI Latin American Congress of Anaesthesiology (WFSA)*.  
Information: Dra M.B. de Azevedo, Rua Paulo Barreto 60, Botafogo, CEP 22280 Botafogo, Rio de Janeiro, RJ, Brazil.
- 6-8 September.** Texas. *Texas Society of Anesthesiologists*.  
Information: Texas Society of Anesthesiologists, 1905 North Lamar Boulevard, # 107, Austin, Texas 78705, USA.
- 11-13 September.** Harrogate. *Linkman and Annual Scientific Meeting of Association of Anaesthetists of Great Britain and Ireland*.  
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 11-15 October.** Baghdad. *4th Pan-Arab Congress of Anaesthesia and Intensive Care*.  
Information: Dr M. Keilani, P.O. Box 17078, Amman, Jordan.
- 26-30 October.** San Francisco. *American Society of Anesthesiologists Annual Meeting*.  
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 6-9 November.** Kuala Lumpur. *7th Asian Congress of Anaesthesiologists*.  
Information: Dr S.W. Lim, Pantai Medical Centre, 59199 Kuala Lumpur, Malaysia.
- 8-10 November.** Vina del Mar. *2nd Congress of Fed. of South American Soc. of Anesthesiologists*.  
Information: Dr Guillermo Lema, Av. Providencia 1476 (Depto. 405) Santiago, Chile.
- 8-11 November.** Toronto. *Paediatric Anaesthesia Conference*.  
Information: Sheila M. Peart, Paediatric Anaesthesia Conference, The Hospital for Sick Children, 555 University Avenue, Toronto, Ont M5G 1X8.
- 1-4 December.** Bangkok. *6th Congress of Western Pacific Association of Critical Care Medicine*.  
Information: Dr P. Sakolsatayadorn, Surgery, Siriraj Hospital, Bangkok 10700, Thailand.
- 6-8 December.** Washington. *Washington State Society of Anesthesiologists Annual Meeting*.  
Information: Washington State Society of Anesthesiologists, 2033 Sixth Avenue, # 804, Seattle, Washington 98121, USA.
- 7-11 December.** New York. *Forty-fifth Postgraduate Assembly in Anesthesiology*.  
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

1992

- 1-8 February.** Colorado. *18th Annual Vail Conference in Anaesthesiology*.  
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101, USA.
- 13-17 March.** San Francisco. *66th Congress of the International Anesthesia Research Society*.  
Information: International Anesthesia Research Society, 3645 Warrenville Center Road, Cleveland, Ohio 44122, USA.
- 25-29 March.** Tampa. *17th Annual Meeting of the American Society of Regional Anesthesia*.  
Information: P.O. Box 11086, Richmond, Virginia, 23230-1086, USA.
- 29 March-2 April.** Atlanta, Georgia. *The Third International Symposium on the History of Anaesthesia*.  
Information: R.K. Calverley, Medical Center, University of California, 225 Dickinson Street, San Diego, California CA 92103, USA.
- 1-3 April.** Bristol. *Junior Anaesthetists' Group Linkman Conference and Annual Scientific Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 2-6 May.** Boston. *Society of Cardiovascular Anesthetists*.  
Information: P.O. Box 11086, Richmond, Virginia, 23230-1086, USA.
- 4-9 June.** Toronto. *49th Annual Meeting of the Canadian Anaesthetists' Society*.  
Information: 187 Gerrard Street E, Toronto, Canada M5A 2E5.
- 7-12 June.** Barcelona. *Anesthesia 92*.  
Information: Pacifico, S.A.: c/Muntaner, 112 08036-Barcelona, Spain.
- 10-13 June.** Brussels. *European Society of Regional Anaesthesia (UK) Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 12-19 June.** The Hague. *10th World Congress of Anaesthesiology*.  
Information: Dr Harm Lip, Nilantweg, 99, 8041 AR Zwolle, Netherlands.
- 9-11 September.** Bournemouth. *Linkman and Annual Scientific Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 17-21 October.** New Orleans. *American Society of Anesthesiologists Annual Meeting*.

*Information:* Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.

**12-16 December.** New York. *46th Postgraduate Assembly in Anesthesiology.*

*Information:* Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists, Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

### 1993

**12-16 February.** Utah. *38th Annual Postgraduate Course in Anesthesiology — 'Anesthesiology: Today and Tomorrow'.*

*Information:* Vicky Larson, Department of Anesthesiology, University of Utah School of Medicine, 50 North Medical Drive, Salt Lake City, Utah 84132, USA.

**29 April-2 March.** North Carolina. *Meeting of the Association of University Anesthetists.*

*Information:* Francis M. James III, Department of Anesthesia, Wake Forest University Medical Center, 300 S. Hawthorne Road, Winston-Salem, North Carolina 27103, USA.

**1-4 September.** Liverpool. *European Course and Congress in Paediatric Anaesthesia.*

*Information:* Dr P.D. Booker, Alder Hey Hospital, Liverpool L12 2AP.

**22-24 September.** Glasgow. *Linkman Conference and Annual Scientific Meeting.* Joint Meeting between the Association of Anaesthetists of Great Britain and Ireland and the Canadian Anaesthetists' Society.

*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

**9-13 October.** Washington DC. *American Society of Anesthesiologists Annual Meeting.*

*Information:* Executive Secretary, ASA 515 Busse Highway, Park Ridge, IL 60068, USA.

### 1994

**7-9 September.** Brighton. *Linkman Conference and Annual Scientific Meeting.*

*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

**2-7 October.** Jerusalem. *European Congress of Anaesthesiology.*

*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

# Contents: Anaesthesia, vol. 46, no. 1, January 1991

## EDITORIAL

### Tributes and tribulations

*M. Morgan*

1

## ORIGINAL ARTICLES

A comparison of the performance of 20 pulse oximeters under conditions of poor perfusion

*D.G. Clayton, R.K. Webb, A.C. Ralston, D. Duthie and W.B. Runciman*

3

Regional anaesthesia and cough effectiveness. A study in patients undergoing Caesarean section

*A.W. Harrop-Griffiths, A. Ravalia, D.A. Browne and P.N. Robinson*

11

Effect of glyceryl trinitrate and ice on dilation of hand veins

*S. Williams and J.F. Hecker*

14

Influence of temperature of bupivacaine on spread of spinal analgesia

*T. Callesen, I. Jarnvig, B. Thage, T. Krantz and C. Christiansen*

17

Propofol for induction and maintenance of anaesthesia at Caesarean section. A comparison with thiopentone/enflurane

*G. Yau, T. Gin, M.C. Ewart, C.F. Kotur, R.K.W. Leung and T.E. Oh*

20

Ketamine as analgesic for total intravenous anaesthesia with propofol

*J.B.M. Guit, H.M. Koning, M.L. Coster, R.P.E. Niemeijer and D.P. Mackie*

24

## CASE REPORTS

Midazolam-induced benzodiazepine withdrawal syndrome

*B. Mets, A. Horsell and D.M. Linton*

28

Response to suxamethonium in a myasthenic patient during remission

*M. Abel, J.B. Eisenkraft and N. Patel*

30

Emery-Dreifuss muscular dystrophy

*P. Morrison and R.H. Jago*

33

Emergency adenotonsillectomy for acute postoperative upper airway obstruction

*J.R. Livesey, N.B. Solomons and E.A.D. Gillies*

36

Non-drug related asystole associated with anaesthetic induction

*T.K. Keane, P.J. Hennis and M.Th.E. Bink-Boelkens*

38

Rupture of the oesophagus during cricoid pressure

*S.J. Ralph and C.A. Wareham*

40

## APPARATUS

Accidental bronchial intubation with RAE tubes

*A.E. Black and A.M. Mackersie*

42

A comparison of patient rewarming devices after cardiac surgery

*M.M. Imrie, M. Ward and G.M. Hall*

44

## HISTORICAL NOTES

Dr F.P. de Caux—the first user of curare for anaesthesia in England

*D.J. Wilkinson*

49

## FORUM

Minimum oxygen requirements during anaesthesia with the Triservice anaesthetic apparatus. A study of drawover anaesthesia in the young adult

*S.Q.M. Tighe, G.A. Turner, S.B. Merrill and R.J. Pethybridge*

52

The role of a critical care unit in an epidemic

*A. Kumar, S. Gupta, S. Ahuja, A. Sethi and A. Bhattacharya*

57

Factors that influence the induction dose of propofol

*G.J. McCleane, D.F. Fogarty and C.H. Watters*

59

Awareness during Caesarean section

*G. Lyons and R. Macdonald*

62

Coagulation screening before epidural analgesia in pre-eclampsia

*P. Barker and C.C. Callander*

64

Modification of pain on injection of propofol—a comparison between lignocaine and procaine

*M.E. Nicol, J. Moriarty, J. Edwards, D.S. Robbie and R.P. A'Hern*

67

## CORRESPONDENCE

70

## BOOK REVIEWS

78

## OBITUARIES

80

## INTERNATIONAL CONGRESS CALENDAR

80



0003-2409(199101)46:1-W



1292

# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 46    Number 4    April 1991



The Association of Anaesthetists of Great Britain and Ireland  
*Patron: HRH The Princess Margaret, Countess of Snowdon*



Published for the Association by  
Academic Press                      Grune & Stratton  
London    San Diego    New York    Boston  
Sydney    Tokyo    Toronto



ISSN 0003-2409



# SLEEPING BEAUTY

***Isoflurane***

An inspired solution



**Prescribing Information:** Isoflurane. **Indications:** for inhalation anaesthesia. **Dose:** Induction should begin at 0.5% and be adjusted appropriately. Maintenance concentrations generally lie between 1.0% and 2.5%. For caesarian section, 0.5–0.75% Isoflurane in a mixture of oxygen/nitrous oxide is recommended. Elderly: lesser concentrations normally required. **Contraindications:** known sensitivity to Isoflurane or a history of malignant hyperpyrexia following a previous administration. **Side effects:** hypotension and respiratory depression. Shivering, nausea and vomiting may occur during recovery. **Basic N.H.S. price:** £33.50 per 100ml. **PL Number:** 0037/0115. Further information is available upon request from: **Abbott Laboratories Limited**, Abbott House, Moorbridge Road, Maidenhead.

**ABBOTT**



# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

---

Editor M. MORGAN

Assistant Editors R. GREENBAUM, A. R. AITKENHEAD, D. E. F. NEWTON, R. A. MASON, M. HARMER

## *Editorial Board*

Chairman The President of the Association P. J. F. BASKETT

Members A. M. ROLLIN, C. A. B. MCLAREN and a representative of the Junior Anaesthetists' Group

## *Ex officio*

The Honorary Treasurer of the Association W. R. MACRAE

The Honorary Secretary of the Association J. E. CHARLTON

The Immediate Past Honorary Secretary of the Association W. L. M. BAIRD

---

DIANE WINTERBURN, JANET SPRINGER, Reference librarians

---

*Address for literary contributions* Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK [telephone 081-740 3290].

Neither the Editor nor the Association of Anaesthetists of Great Britain and Ireland is in any way responsible for the statements made or the views expressed by contributors.

Investigations performed on man must conform to appropriate ethical standards including voluntary, informed consent and acceptance by an ethics committee.

A written statement of this acceptance may be requested by the Editor.

---

ANAESTHESIA: ISSN 0003-2409, Volume 46 1991, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. All advertising enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293). Annual subscription price including postage: £132 UK and US \$244 overseas. Personal subscription rate including postage: \$99. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel: 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

*Anaesthesia* is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

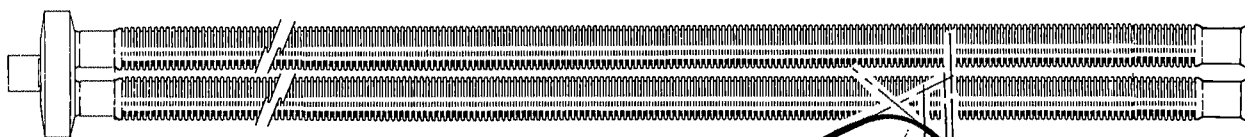
© 1991 The Association of Anaesthetists of Great Britain and Ireland. The appearance of the code at the bottom of the first page of a paper in this journal indicates the copyright owner's consent that copies of the paper may be made for personal or internal use, or for the personal or internal use of specific clients in the USA. This consent is given on the condition, within the USA, that the copier pay the stated per-copy fee through the Copyright Clearance Center Inc., 27 Congress Street, Salem, MA 01970, USA for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to other kinds of copying, such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, for resale or for copying or distributing copies outside the USA.

Origination by BPCC Whitefriars Ltd, Tunbridge Wells, and printed and bound in Great Britain by BPCC Wheatons Ltd, Exeter.



# The safer alternative

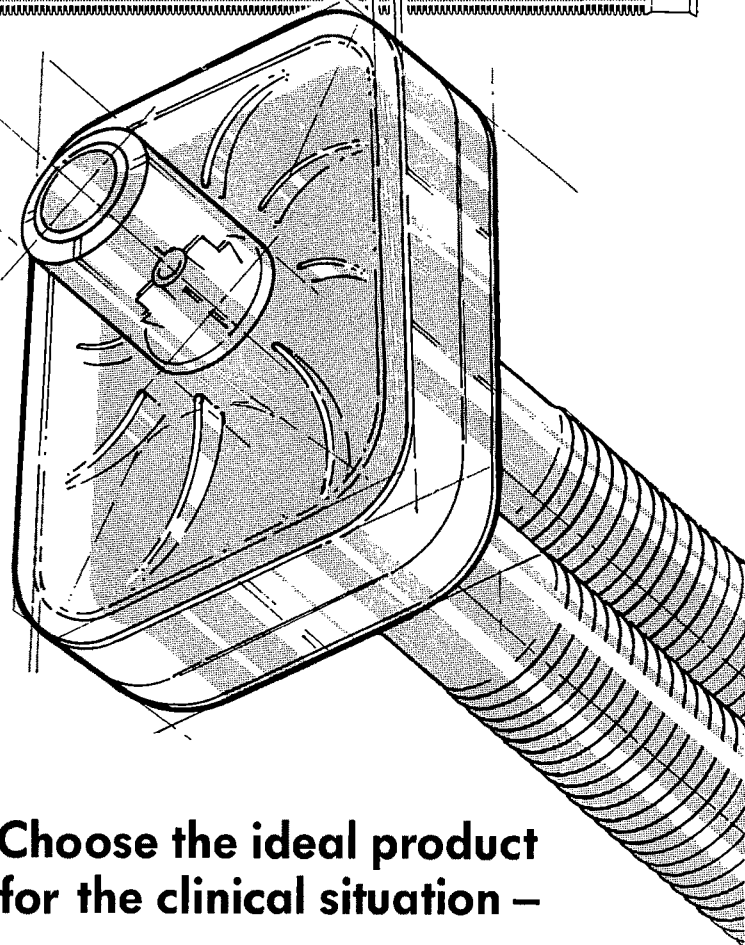
## Filtration Technology at its finest



**A range of unique Breathing Filters/HME's to resolve all your filtration and humidification problems**

The Filta-Therm – providing both Heat and Moisture Exchange and Bacterial/Viral Filtration 99.999% – ideal for **patient protection and humidification.**

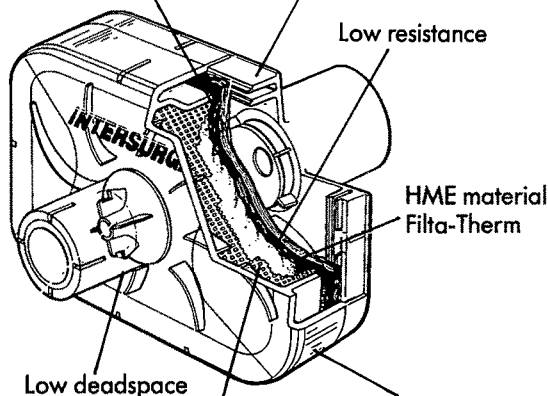
The Filta-Guard – providing Bacterial/Viral Filtration 99.999% – ideal for **equipment protection – no wastage.**



Filter matrix  
99.999% efficiency

Light-weight

Low resistance



HME material  
Filta-Therm

Low deadspace

If mismanaged  
will not block

Smooth profile

**Choose the ideal product for the clinical situation –**

**Choose a safe product – choose Intersurgical.**

**INTERSURGICAL**  
COMPLETE RESPIRATORY SYSTEMS

Crane House, Gould Road,  
Twickenham, Middlesex TW2 6RS.  
Tel: 081-755 1477, Fax: 081-755 2177



G.M.P Registered  
No. RO252/ME

I am interested in further details of Filta-Guard ☐ Filta-Therm ☐  
Intersurgical, Crane House, Gould Road, Twickenham, Middlesex TW2 6RS.

Name

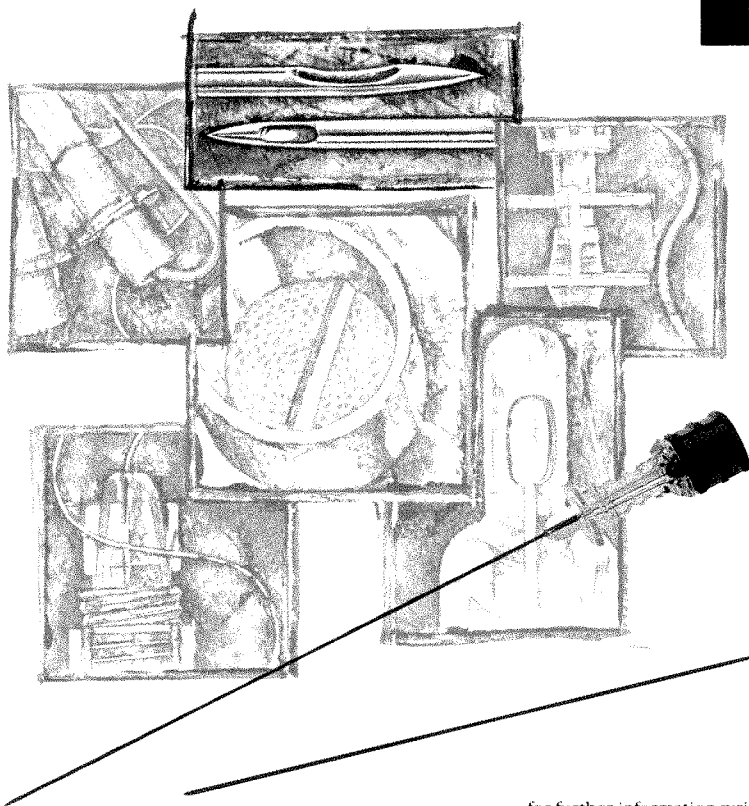
Position

Dept.

Hospital

Hospital Address

Equipment used



## The Sprotte Spinal Needle

### The new atraumatic spinal needle

- Atraumatic tip  
displaces dural fibres
- Dural tap closes on  
withdrawal of needle
- Minimises loss of CSF  
and subsequent post  
dural puncture headaches
- Easy injection and  
aspiration due to  
the relatively wide  
lateral eye

21g, 22g, 24g

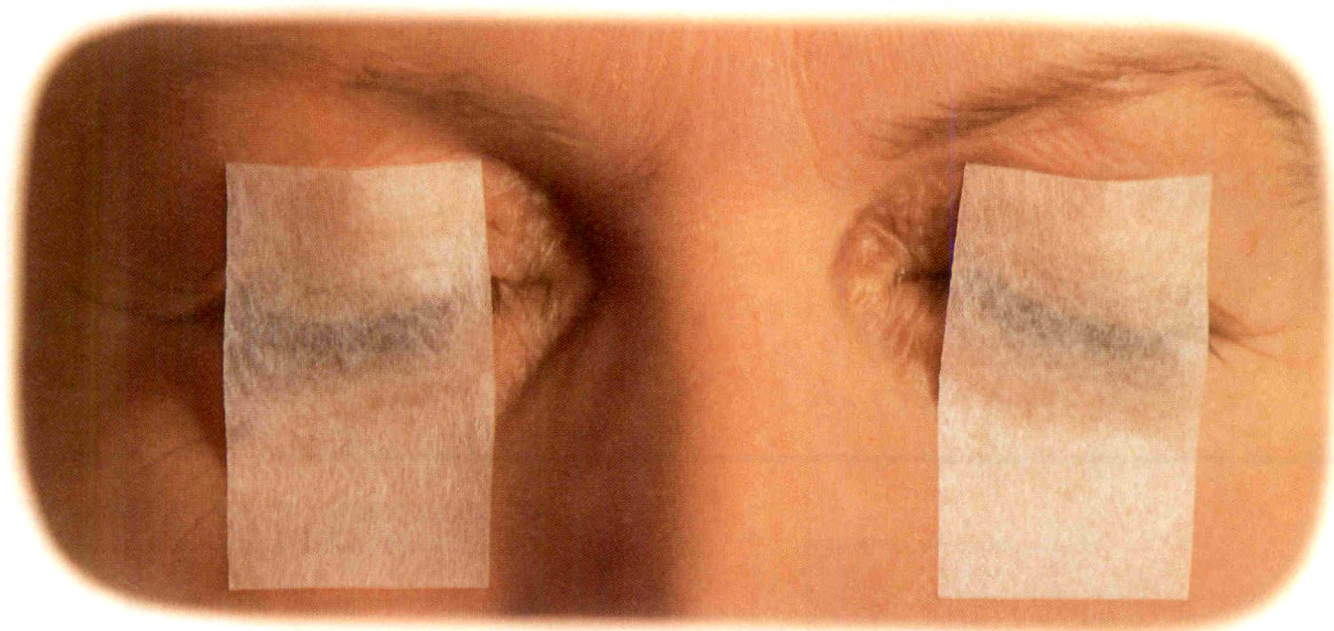
Sprotte spinal needles

now available from:

for further information write to:  
RUSCH UK Ltd. Freepost, High Wycombe, Bucks. HP12 3BR or Freephone 0800 581121

**ITI RUSCH** UK Ltd.

# HOW CAN A HIP OPERATION AFFECT THE EYE?



Basal tear production can decline by as much as 95% during general anaesthesia,<sup>1</sup> resulting in drying of the corneal epithelium and painful corneal abrasions.<sup>2</sup>

That's why it's so important to protect the cornea with LACRI-LUBE, the leading ophthalmic ointment for the treatment of dry eye.

LACRI-LUBE supplements deficient tears throughout a surgical procedure, helping to prevent accidental tape adhesion to the cornea or conjunctiva. In long procedures, further applications may be required.



And now LACRI-LUBE is available in a new **preservative-free** formulation which eliminates the potential for preservative sensitivities.

Yet another reason for you to choose LACRI-LUBE as an automatic part of your induction routine. After all, who needs eye problems after a hip operation?



## LACRI-LUBE\*

(preservative free ophthalmic ointment)

FOR CONTINUOUS CORNEAL PROTECTION DURING  
GENERAL ANAESTHESIA

#### LACRI-LUBE\*

**Presentation:** Off-white, smooth, preservative-free sterile ophthalmic ointment containing white petrolatum, mineral oil and non-ionic lanolin derivatives. **Uses:** Useful as adjunctive therapy to lubricate and protect the eye in conditions such as exposure keratitis, decreased corneal sensitivity, recurrent corneal erosions, keratitis sicca, and also in ophthalmic and non-ophthalmic surgery. **Dosage and Administration:** For topical administration. Pull lower lid down to form pocket and apply a small amount as required. There is no variation of dose for age. **Contraindications, warnings, etc.** No known contra-indications. **Pharmaceutical precautions:** Store away from heat. To avoid contamination during use, do not touch tube tip to any surface. **Legal Category:** P. **Package Quantities:** Lacri-Lube is available in 3.5g and 5g ophthalmic ointment tubes.

\*Trade Mark.

**Basic NHS cost:** 3.5g £1.97, 5g £2.49 (as at January 1991). **Further Information:** Dry eye symptoms commonly persist at night - Lacri-Lube has been specifically formulated to lubricate and protect the dry eye during sleep. Lacri-Lube can provide prophylactic ocular care during general surgical procedures as an adjunct to taping of the eyelids. **Product licence number:** 0426/0041

 **ALLERGAN PHARMACEUTICALS**

Allergan Ltd., High Wycombe, Bucks HP12 3SH.

**References:** 1. Cross, D.A. et al., *Anesthesia and Analgesia* ... *Curr Res* 1977; **56** (1): 35-37. 2. Snow, J.C. et al., *Anesthesia and Analgesia* ... *Curr Res* 1975; **54** (4): 465-467.



## Editorial

### Ethics in publishing

In January 1978, editors from some major biomedical journals published in English met in Vancouver, British Columbia, and drew up technical requirements for manuscripts to be submitted to these journals. The Vancouver group evolved into the International Committee of Medical Journal Editors (ICMJE), which in 1981 revised the requirements and published a second edition in 1982, and a third edition in 1988.<sup>1</sup> While the *Uniform Requirements* describe mainly the way in which a manuscript should be prepared, they also make important recommendations on some ethical aspects: notably prior and duplicate publication, authorship, financial relationships posing conflict of interests, and requirements that procedures were conducted in accordance with the ethical standards of the appropriate committee on human experimentation or with the Helsinki Declaration.

More specifically with respect to duplicate publication, in the *Uniform Requirements* it is stated that most journals do not wish to consider for publication a paper describing work that has already been reported elsewhere or that has been submitted or accepted for publication elsewhere. (This does not preclude considering a paper that has been rejected by another journal or a complete report of a preliminary communication.) When submitting a paper, an author should always make a full statement about all submissions and previous reports that might be regarded as prior or duplicate publication of the same or very similar work and copies of such material should be included with the submitted paper.

As the *Uniform Requirements* are not immediately available to most authors, many journals including *Anesthesiology*, *Anesthesia and Analgesia*, *Anaesthesia* and *British Journal of Anaesthesia*, publish an individual Guide to Contributors which summarizes these findings and describes specific requirements. In our Guide to Contributors, it is stated clearly that papers submitted must not have been submitted to or published *in whole* or *in part* in any other journal. Despite these well publicised strictures, there have been several examples in the past 2 years wherein material published in or submitted to one of our journals has been either simultaneously submitted to or published in a second anaesthesia journal.

An example of duplicate publication which authors may think acceptable but which we invariably reject, comprises the use of control data to compare with one situation in one journal and the same control data compared against a second circumstance in a paper submitted to another journal. When authors fail to indicate in their list of references that material is 'in press', or published recently in another journal, one conclusion is that the authors are deliberately practising deception.

For the past 8 years, an International Meeting of Editors of Anaesthetic Journals has been convened at 2-year intervals coinciding with either a European or World Congress of Anaesthesiology. At the most recent

Fourth Meeting held in association with the Eighth European Congress of Anaesthesiology in Warsaw in 1990, there was some discussion of ethical problems in publishing. The most common and obvious problem discussed was that of duplicate submission and publication. This may be classified broadly into two types:

(1) Reproduction in one journal of identical data to that published elsewhere with either similar or differing discussions. This is clearly unacceptable and represents a breach of copyright. Authors should be aware of the fact that reproduction of *blocks* of text, tables or figures always necessitates permission of both authors and editors.

(2) The use of identical control data in two different papers, as noted above.

An additional issue of concern is the submission of various aspects of one single study to several different journals. Although this does not represent multiple publication *per se*, it nonetheless represents publication of an unnecessary number of papers and detracts from the interpretation of single manuscripts. All the editors present at the Fourth International Meeting of Editors of Anaesthetic Journals in *Warsaw*, 1990, were opposed to this type of practice and would request that all data from a single circumscribed study be submitted to one journal.

This international collaboration is illustrated by the fact that this joint editorial is appearing simultaneously in *Anesthesiology*, *Anesthesia and Analgesia*, *British Journal of Anaesthesia* and *Anaesthesia*. The views expressed on duplicate publication are also supported fully by David Bevan, who is publishing a similar editorial in this month's issue of the *Canadian Journal of Anaesthesia*, and also by John Roberts, who will be referencing this article in an editorial in *Anaesthesia and Intensive Care*.

Improving communication between the editors of biomedical journals should facilitate detection of any failure of authors to follow the recommendations of the ICMJE and we hope that authors become aware of and exercise their responsibilities related to submission of manuscripts. Research published in biomedical journals must be held to the highest standards and adherence to ethical standards in publishing is an absolute expectation of our readers as well as the public.

Editor,  
*British Journal of Anaesthesia*  
Editor in Chief,  
*Anesthesia and Analgesia*  
Editor in Chief,  
*Anesthesiology*  
Editor,  
*Anaesthesia*

G. SMITH

R. MILLER

J. SAIDMAN

M. MORGAN

### Reference

1. International Committee for Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. *British Medical Journal* 1988; **296**: 401-5.

### Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; **1**: 432-5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

## Double-blind comparison of epidural diamorphine and intramuscular morphine after elective Caesarean section, with computerised analysis of continuous pulse oximetry

J. D. STEVENS, P. BRAITHWAITE, C. F. CORKE, T. H. MADEJ AND R. G. WHEATLEY

### Summary

*A randomised, double-blind comparison of the efficacy, duration of action and side effects of two analgesic regimens following elective epidural Caesarean section is described. Patients received epidural diamorphine 3 mg or intramuscular morphine 10 mg in the immediate postoperative period. Time to next analgesia was longer after epidural diamorphine (11.0 hours) compared to intramuscular morphine (6.5 hours) ( $p < 0.05$ ). In addition, a greater number of patients in the diamorphine group had a pain score  $< 2.5$  cm at 5 hours ( $p < 0.05$ ). However, more patients in the diamorphine group required catheterisation and suffered emetic sequelae, whereas more patients in the morphine group were sedated at 8 hours. Ten patients in each group had continuous pulse oximetry performed overnight after administration of the trial medications. Neither group demonstrated evidence of hypoxia.*

### Key words

*Analgesics; diamorphine, morphine.*

*Anaesthetic techniques, regional; epidural.*

*Oxygen; measurements.*

The effect of local anaesthetic gradually diminishes after Caesarean section performed under epidural anaesthesia. Thereafter, pain is experienced which almost always necessitates the administration of opioid analgesics. The epidural route was used in this situation.<sup>1–4</sup> Access can be gained via the epidural catheter sited to establish regional nerve block with local anaesthetic. Epidural opioids offer potential advantages of increased efficacy, duration of action and reduced side effects compared to other routes of administration.<sup>1</sup>

Diamorphine has a high degree of lipid solubility, a property which appears related to the efficacy of opioids when administered by the epidural route.<sup>2</sup> In particular, epidural lipophilic opioids should have a fast onset of action and a reduced potential for delayed respiratory depression.<sup>3</sup>

Epidural diamorphine was shown to have prolonged duration of action compared to the same dose given intramuscularly.<sup>4–6</sup> The doses used in these studies were 5 mg, 0.1 mg/kg and 5 mg respectively. A 5-mg dose of diamorphine epidurally has resulted in an unacceptable

incidence of side effects in combination with bupivacaine during labour.<sup>7</sup> Study of a lower dose of 3 mg has been recommended. Our standard dose of epidural diamorphine after lower abdominal surgery is 3 mg.<sup>8</sup> A satisfactory, controlled, double-blind study of low-dose epidural diamorphine (less than 0.05 mg/kg) for postoperative pain relief has not been performed.

This study was designed to determine whether a single bolus of epidural diamorphine 3 mg had any advantages compared to starting conventional intramuscular morphine 10 mg immediately following elective Caesarean section under epidural anaesthesia.

### Methods

Approval was obtained from the hospital Medical Ethics Committee. Written, informed consent was obtained from 44 (ASA 1 and 2) parturients scheduled for elective Caesarean section under epidural anaesthesia.

Routine preparation included one dose of intramuscular metoclopramide given one hour before transfer to the

J.D. Stevens,\* MB, BS, FFARCSI, P. Braithwaite,† MB, ChB, FFARCSI, C.F. Corke,‡ BSc, MB, BS, MRCP, FFARCS, Registrars, T.H. Madej,\* BM, FFARCS, R.G. Wheatley,\* MB, ChB, FFARCS, Consultants, York District Hospital, Wigginton Road, York YO3 7HE.

Current addresses: \*York District Hospital; †Killingbeck Hospital, York Road, Leeds LS14 6UH, ‡Repatriation General Hospital, Heidelberg, Melbourne, Australia.

Accepted 13 August 1990.



delivery suite and prophylactic antacid therapy. Patients had an epidural nerve block established using 0.5% plain bupivacaine given via a lumbar epidural catheter, and a sensory block was produced from S<sub>2</sub>-T<sub>4</sub>. Up to 2 litres of crystalloid solution was infused prophylactically to maintain the arterial blood pressure above 100 mmHg systolic, and ephedrine was used in addition, as required. No opioid supplement or anticholinergic was administered during the Caesarean section, and any peroperative discomfort was treated with 35–50% nitrous oxide in oxygen.

At conclusion of surgery (time zero), patients received two injections, 10 ml epidurally and 2 ml intramuscularly. One injection contained opioid, either epidural diamorphine 3 mg or intramuscular morphine 10 mg, while the other was a placebo of normal saline. The solutions were prepared by the hospital pharmacy and allocation was randomised and double-blind. The epidural injection was given via the lumbar epidural catheter sited to establish regional anaesthesia and the intramuscular injection was given into an anaesthetised thigh. All patients had their epidural catheter removed after the injections were administered. No antiemetic was given with the study solutions.

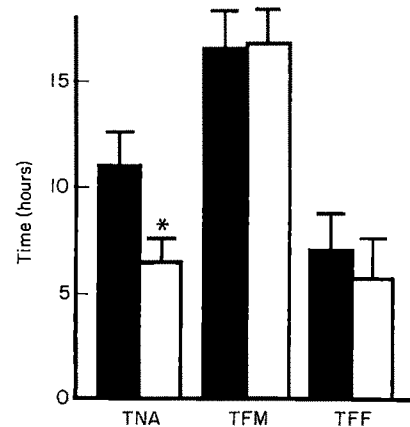
Any additional analgesia was provided on request from the patient and given by the nursing staff as intramuscular morphine 10 mg combined with an antiemetic. The number of hours between administration of the injections to the first additional dose of postoperative analgesia was termed the time to next analgesia.

Patients recorded their pain scores using a 10-cm visual analogue scale<sup>9</sup> hourly for 8 hours. At 4, 8 and 24 hours postoperatively, the patients were visited by an anaesthetist and asked specifically about mobilisation, feeding the baby and emetic sequelae (graded: 0, no sickness; 1, nausea; 2, vomiting). The degree of sedation was determined by a four-point rank sedation score (graded: 1, awake; 2, drowsy; 3, very drowsy; 4, asleep).<sup>10</sup> Any requirement for urinary catheterisation was documented. All patients had peak flows measured at time 0, 4 and 8 hours. These were used to demonstrate any deterioration in respiratory function due to pain or sedation. Twenty patients (10 in each group) had continuous arterial oxygen saturation measured with a Nellcor N-100 pulse oximeter interfaced with an Opus, IBM-compatible microcomputer.<sup>8,11</sup> The oximeter probe was attached to the patient's great toe. Recordings of SaO<sub>2</sub> were made every 10 seconds and stored by the computer so that 360 samples were obtained every hour. The display and analysis of these data have already been described.<sup>8</sup> Oximetry was performed on admission to the postnatal ward over the first postoperative night. On the seventh postoperative night, oximetry was repeated with the patients acting as their own drug-free control.

Results were subjected to statistical analysis by Student's *t*-test or Chi-squared test with Yates' correction as appropriate. The visual analogue scores (VAS) for pain were

**Table 1.** Clinical data of investigated patients (mean, SEM) (Student's *t*-test).

Analgesic regimen	Age; years	Bupivacaine dose; mg
Epidural diamorphine ( <i>n</i> = 22)	27.6 (1)	120 (5)
Intramuscular morphine ( <i>n</i> = 22)	29.8 (1.1)	114 (4)



**Fig. 1.** Mean (SEM) time to next analgesia (TNA), first mobilisation (TFM) and first feeding (TFF). \*Statistical significance ( $p < 0.05$ ). ■, epidural diamorphine; □, intramuscular morphine.

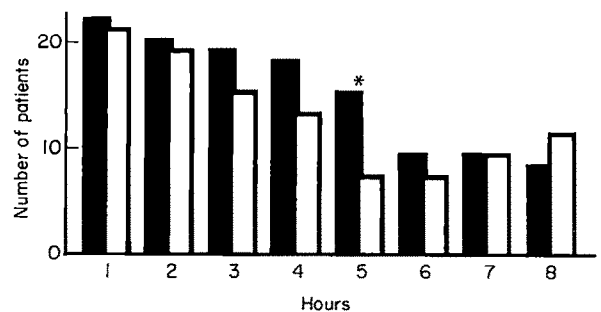
treated as an ordinal scale as follows: 0–2.4 cm nil or just noticeable; 2.5–4.9 cm, mild; 5.0–7.4 cm, moderate; 7.5–10.0 cm, severe. Results of the pain scores were analysed nonparametrically.<sup>12</sup>

## Results

The mean ages and dose of bupivacaine for epidural anaesthesia are shown in Table 1. There was no significant difference between the groups.

Nine patients required nitrous oxide supplementation, six subsequently received diamorphine and three morphine. Figure 1 shows the mean time to next analgesia, time to first mobilisation and time to first feeding of the baby. The time to next analgesia was significantly longer in the epidural diamorphine group than the intramuscular morphine ( $p < 0.05$ ). However, there were no significant differences between time to first mobilisation and first feeding the baby. Five patients in the diamorphine group and one in the morphine group required no further doses of postoperative analgesia.

The mean number of additional doses of postoperative analgesia (SEM) were 1.8 (0.2) for the diamorphine group, and 2 (0.2) in the morphine group. There was no statistically significant difference between the groups in the amount of postoperative analgesia required. The number of patients with VAS for pain less than 2.5 cm at 1 to 8 hours are shown in Figure 2. At 5 hours, the diamorphine group had significantly larger numbers of patients with



**Fig. 2.** Number of patients with visual analogue score (VAS) for pain < 2.5 cm. \*Statistical significance ( $p < 0.05$ ). Symbols as for Figure 1.

Table 2. Number of patients with side effects.

Side effect	Epidural diamorphine (n=22)	Intramuscular morphine (n=22)
Emetic sequelae (nausea ± vomiting) at:		
4 hours	6	4
8 hours	9	2*
Antiemetic administration	15	13
Sedation (grades 2, 3, or 4)† at:		
4 hours	17	21
8 hours	16	22*
Catheterisation	12	4*

\*Statistical significance by Chi-squared test ( $p < 0.05$ ).

†Rank Sedation Score.

VAS less than 2.5 cm compared to the morphine group ( $p < 0.05$ ).

Side effects of the treatments are shown in Table 2. The diamorphine group had significantly more patients with emetic sequelae (nausea or vomiting) at 8 hours. More patients in the diamorphine group required urinary catheterisation ( $p < 0.05$ ). All patients in the morphine group were sedated with rank sedation scores of 2, 3, or 4 at 8 hours ( $p < 0.05$ ).

Peak flows measured at 0, 4 and 8 hours in each group showed no significant difference between or within groups at any time.

Table 3 shows the result of the computerised analysis of continuous pulse oximetry for the first postoperative night (with drug) and seventh postoperative night (control), in both groups. There was no evidence of hypoxia compared to control in either group.

Figure 3 (a and b) shows examples of typical graphical representations, compressed spectral arrays of continuous pulse oximetry results for two different patients.<sup>11</sup> The hourly peak values represent the modal value of  $SaO_2$  for each hour of monitoring. Figure 3(a) demonstrates non-hypoxaemic unstable and Figure 3(b) nonhypoxaemic stable patterns of  $SaO_2$ . Hypoxaemia is defined as an  $SaO_2 < 94\%$  for more than 6 minutes/hour and an unstable pattern is where the variation between the hourly peaks is greater than 4%.<sup>8</sup>

Table 4 shows the numbers of patients in each group (control versus drug) with nonhypoxaemic unstable

Table 3. Analysis of pulse oximetry. Number of minutes/hour  $SaO_2 < 94\%$  (mean, SEM).

	Epidural diamorphine (n=10)	Intramuscular morphine (n=10)
Control (7th night)	1.1 (0.5)	0.6 (0.3)
With drug (1st night)	0.6 (0.2)	0.9 (0.5)

Table 4. Analysis of pulse oximetry. Number of patients with nonhypoxaemic unstable pattern of  $SaO_2$ .

	Epidural diamorphine (n=10)	Intramuscular morphine (n=10)
Control: (7th night)	4	6
With drug: (1st night)	3	9*

\*Statistical significance calculated using Student's *t*-test ( $p < 0.05$ ).

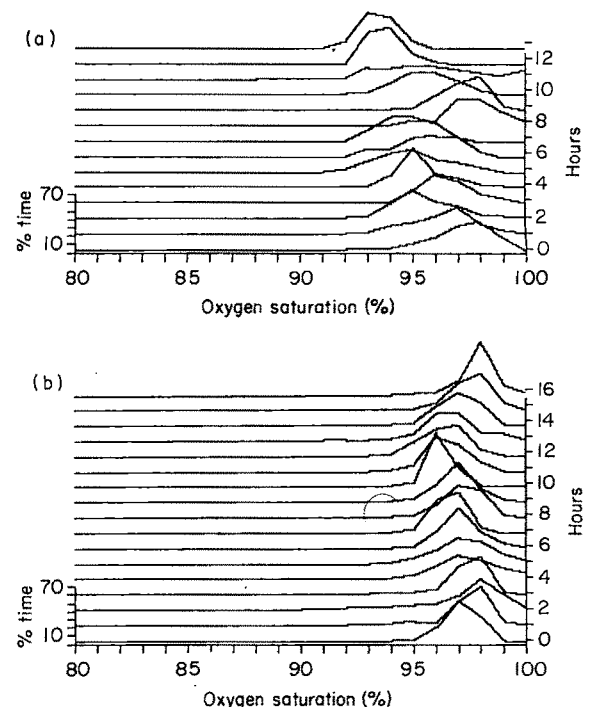


Fig. 3. Compressed spectral arrays of typical patients showing nonhypoxaemic unstable (a) and stable (b) patterns of arterial oxygen saturation ( $SaO_2$ ).

patterns of  $SaO_2$ . More patients in the morphine group had a nonhypoxaemic unstable pattern of  $SaO_2$  ( $p < 0.05$ ).

### Discussion

The main aim of our study was to improve existing methods of postoperative analgesia after elective epidural Caesarean section in a safe and efficient manner. It was not possible in a busy district general hospital to offer on-demand bolus epidural analgesia on the postnatal wards. Therefore the study was designed to establish whether a single bolus dose of epidural diamorphine 3 mg offered an advantage compared to starting conventional intramuscular morphine 10 mg immediately after operation. The drug dose chosen was that routinely recommended and used, as the potency ratio for epidural and systemic administration is unknown.

We have demonstrated that epidural diamorphine 3 mg was superior in duration of analgesia compared to intramuscular morphine 10 mg: the epidural diamorphine group had more patients with low pain scores at 5 hours. At this point the morphine group were 90 minutes short of their mean time to next analgesia and the effect of the initial intramuscular dose was probably receding. Low dose epidural diamorphine and morphine have been shown to give better analgesia compared to intramuscular opioids after lower abdominal surgery.<sup>8,13</sup> However, administration was not randomised or double-blind in these studies. Other workers have failed to demonstrate that low dose epidural opioids provide adequate analgesia in the early postoperative period.<sup>1,5</sup> A lower abdominal incision, early prophylactic administration, a synergistic effect with residual local anaesthetic and psychological factors associated with parturition could have contributed to the efficacy of both regimens in our study.

Nausea and vomiting were grouped together and given

equal weight in the analysis of emetic sequelae, since both can be equally distressing to the patient. We demonstrated an increased incidence of emetic sequelae at 8 hours in the diamorphine group. More patients in this group required perioperative nitrous oxide supplementation. However, we do not feel that this is likely to be the causative factor. Nitrous oxide was administered for short periods, at low concentrations and without intermittent positive pressure ventilation. In addition, emetic sequelae due to nitrous oxide would be expected early in the postoperative period and not at 8 hours. We feel that a more likely explanation for the disparity between the groups in the incidence of emetic sequelae at 8 hours is due to routine prophylactic antiemetic administration with the first additional postoperative dose of morphine, which was requested earlier in the morphine group. The mean time to next analgesia in the morphine group was 6.5 hours compared to 11 hours in those who received diamorphine. The timing of the first additional dose of intramuscular morphine and the concurrent administration of an antiemetic could also explain why all the patients in the morphine group were sedated at 8 hours. Fewer patients in the diamorphine group were sedated at this time. Nonpharmacological effects on sedation were minimised by recruiting elective cases only to the study. The rank sedation score used is a subjective assessment of drowsiness and not anxiolysis.

More patients required urinary catheterisation in the diamorphine group. This has been reported by other workers<sup>14</sup> as a problem of epidural opioids.

We did not ask patients specifically about pruritus and none spontaneously mentioned this symptom or requested treatment. If direct questioning is used the incidence of pruritus after 5 mg of epidural diamorphine has been documented as over 50%.<sup>4,7</sup> However, the pruritus is usually not a troublesome symptom and only rarely requires treatment. No other side effects were volunteered in open questioning.

Large dose epidural opioids with both low and high lipid solubility have resulted in respiratory depression.<sup>15,16</sup> It appears this problem may be significantly reduced if low doses of epidural opioids with high lipid solubility are used. In our study, there was no evidence of respiratory depression as determined by computerised analysis of continuous pulse oximetry.<sup>11</sup> This highly sensitive continuous monitor has been successfully used in the diagnosis of sleep apnoea<sup>17</sup> and to demonstrate hypoxia in other postoperative patients.<sup>8</sup>

Peak flow is a measure of respiratory function, which may be influenced by changes in expiratory force and airway resistance and the effects of pain and the patient's degree of sedation. We used peak flow as a crude measurement of adequate analgesia with minimal sedation at 4 and 8 hours. At time zero the patients had no pain since any perioperative visceral discomfort requiring nitrous oxide supplementation had resolved. The time zero measurement was used as a baseline for expiratory force and airway resistance; the patients had not required any opioid medication, but we appreciate that it could have been influenced by the residual epidural nerve block with local anaesthesia. There was no significant difference between or within the groups in their peak flows at any time.

Portable apparatus for continuous measurement, storage and analysis of pulse oximetry was being developed during this study at York District Hospital.<sup>11</sup> It was available during the latter half of the study and was used on the last

20 patients (10 from each group). The pulse oximetry data did reveal more patients in the morphine group with an unstable pattern of arterial oxygen saturation.<sup>8</sup> At present the significance of this observation is unknown, although it may represent a dampening of central respiratory control and/or maintenance of the upper airway tone.<sup>17</sup>

In conclusion, we have found that low dose epidural diamorphine 3 mg is superior in duration of analgesia compared to conventional intramuscular morphine 10 mg, but at the expense of a higher degree of emetic and urinary side effects.

### Acknowledgments

The authors thank all the nursing and medical staff of the obstetric unit and the pharmacy at the York District hospital for their cooperation, and the anaesthetic research nurse for her assistance.

### References

1. BROMAGE PR, CAMPORESI EM, CHESTNUT D. Epidural narcotics for postoperative analgesia. *Anesthesia and Analgesia* 1980; **59**: 473-80.
2. HOULTON PG, REYNOLDS F. Epidural diamorphine and fentanyl for postoperative pain. *Anaesthesia* 1981; **36**: 1144-5.
3. COUSINS MJ, MATHER LE. Intrathecal and epidural administration of opioids. *Anesthesiology* 1984; **61**: 276-310.
4. MACRAE DJ, MUNISHANKRAPP S, BURROW LM, MILNE MK, GRANT IS. Double-blind comparison of the efficacy of extradural diamorphine, extradural phenoperidine and I.M. diamorphine following Caesarean section. *British Journal of Anaesthesia* 1987; **59**: 354-9.
5. JACOBSON L, PHILLIPS PD, HULL CJ, CONACHER ID. Extradural versus intramuscular diamorphine. A controlled study of analgesic and adverse effects in the post-operative period. *Anaesthesia* 1983; **38**: 10-8.
6. MALINS AF, GOODMAN NW, COOPER GM, PRYS-ROBERTS C, BAIRD RN. Ventilatory effects of pre- and postoperative diamorphine. A comparison of extradural with intramuscular administration. *Anaesthesia* 1984; **39**: 118-25.
7. MCGRADY EM, BROWNHILL DK, DAVIS AG. Epidural diamorphine and bupivacaine in labour. *Anaesthesia* 1989; **44**: 400-3.
8. WHEATLEY RG, SOMERVILLE ID, SAPSFORD DJ, JONES JG. Postoperative hypoxaemia: comparison of extradural, I.M. and patient-controlled opioid analgesia. *British Journal of Anaesthesia* 1990; **64**: 267-75.
9. HUSKISSON EC. Measurement of pain. *Lancet* 1974; **2**: 1127-31.
10. SEMPLE AJ, MACRAE DJ, MUNISHANKRAPP S, BURROW LM, MILNE MK, GRANT IS. Effect of the addition of adrenaline to extradural diamorphine analgesia after Caesarean section. *British Journal of Anaesthesia* 1988; **60**: 632-8.
11. WHEATLEY RG, SOMERVILLE D, JONES JG. Computerized analysis of continuous pulse oximetry for prolonged perioperative studies. *British Journal of Anaesthesia* 1988; **61**: 110P-1P.
12. SCOTT J, HUSKISSON EC. Graphic representation of pain. *Pain* 1976; **2**: 175-84.
13. WEBSTER NR, LYONS G, MACDONALD R. Sleep and comfort after Caesarean section. *Anaesthesia* 1986; **41**: 1143-5.
14. BROMAGE PR, CAMPORESI EM, DURANT PAC, NIELSEN CH. Nonrespiratory side effects of epidural morphine. *Anesthesia and Analgesia* 1982; **61**: 490-5.
15. GUSTAFSSON LL, SCHILDT B, JACOBSEN KJ. Adverse effects of extradural and intrathecal opiates: report of a nationwide survey in Sweden. *British Journal of Anaesthesia* 1982; **54**: 479-86.
16. BROCKWAY MS, NOBLE DW, SHARWOOD-SMITH GH, McCLURE JH. Profound respiratory depression after extradural fentanyl. *British Journal of Anaesthesia* 1990; **64**: 243-5.
17. HANNING CD. Obstructive sleep apnoea. *British Journal of Anaesthesia* 1989; **63**: 477-88.

## Pulse oximeter probes

### A comparison between finger, nose, ear and forehead probes under conditions of poor perfusion\*

D. G. CLAYTON, R. K. WEBB, A. C. RALSTON, D. DUTHIE AND W. B. RUNCIMAN

#### Summary

*The performances of 10 pulse oximeters using finger probes were compared with the same pulse oximeters using alternative probes (eight finger probes, two nose probes and a forehead probe) in poorly perfused patients. All readings were then compared with directly measured arterial blood oxygen saturations. The mean difference (bias, 'accuracy'), standard deviation (precision) and 'drop out' rate for each pulse oximeter combination was determined. An overall ranking of performance of each pulse oximeter was calculated using five criteria (accuracy, precision, number of readings within 3% of standard, percentage of readings given within 3% of standard, expected overread limit in 95% of cases). Nose and forehead probes performed poorly. Some ear probes performed well compared to some finger probes, but the overall performance of probes in other sites compared to finger probes was worse, ( $p = 0.05$ ). Two of eight ear probes and no nose or forehead probes would be expected to be within 4% of the reference value in 95% of readings. The use of finger probes rather than probes in other sites is recommended in the patient with poor peripheral perfusion.*

#### Key words

*Equipment; pulse oximeters.  
Measurement.*

Continuous pulse oximetry has become widespread during anaesthesia, in the peri-operative period, and in the critically ill, and is the subject of recent comprehensive reviews.<sup>1–3</sup>

It is suggested that ear probes may be more reliable and have a faster response time than finger probes in patients who have poor perfusion.<sup>2</sup> Nose probes are also suggested; the rationale is that this area is supplied by the anterior ethmoidal artery (a branch of the ophthalmic artery, which is a branch of the internal carotid artery) in which pulsation persists for longer in the presence of intense peripheral vasoconstriction than digital arteries.<sup>4</sup>

Patients who have undergone cardiopulmonary bypass have poor peripheral perfusion in the immediate post-operative period, which may cause pulse oximeters to either fail to provide a reading or give a 'low signal quality' warning.<sup>5–7</sup> These patients are thus ideal for studies comparing pulse oximeters and different probe placement sites.

This study was designed to compare, under conditions of poor peripheral perfusion, the accuracy of 10 pulse

oximeters which have both finger and either ear, nose or forehead probes.

#### Methods

##### *Patient selection*

The study was approved by the local Human Ethics Committee. Adult patients were studied 30 minutes to 2 hours after transfer from the operating room to the cardiac surgery recovery ward. All patients had undergone surgery involving cardiopulmonary bypass with hypothermia to 25–31°C, and all were sedated and their lungs ventilated mechanically at the time of the study.

##### *Study design*

The age, sex, operation, rectal temperature, systolic, diastolic, mean blood pressures and drug therapies of each patient were recorded.

The performance of 10 pulse oximeters was studied; these were supplied with both a finger probe and an

D.G. Clayton, BSc, MB, BS, FFARCS, R.K. Webb, MB, BS, FFARCS, Senior Staff Specialists, A.C. Ralston, BSc, BAppSc, Medical Physicist, D. Duthie, MB, BCh, FFARCS, Senior Registrar, W.B. Runciman, BSc(Med), MB, BCh, FFARCS, PhD, Professor and Head, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia, Australia 5000.

Correspondence should be addressed to: Professor W.B. Runciman please.

\*Combined study with Australian Patient Safety Foundation, GPO Box 400, South Australia 5001.

Accepted 6 August 1990.

alternative probe and constitute a subset of the 20 pulse oximeters used in our earlier study.<sup>7</sup> The pulse oximeters were supplied by the manufacturers and were checked for compliance with Australian Standard 3200-1986 on electromedical equipment for use in patient care areas.

Each pulse oximeter probe was placed on a patient's finger, earlobe, nose, or forehead as appropriate and according to the manufacturers' instructions. The earlobe was rubbed with an alcohol swab for 30 seconds before application of the ear probe to improve the local circulation, as recommended by several of the pulse oximeter operation manuals. Systemic arterial pressure was monitored continuously through a radial artery cannula. Pulse oximeter finger probes were applied only to fingers on the arm not bearing the arterial cannula and were not applied to the thumb. The finger used for each manufacturer's probe was varied in a predetermined fashion to ensure that each probe was placed an equal number of times on each of the fingers used and to eliminate any possible systematic interference from adjacent probes. A two-dimensional matrix was constructed after allocating a number to each pulse oximeter and a letter to each finger so that it could be checked visually that these requirements were met. All finger probes were covered by opaque rubber sleeves to reduce the likelihood of crossover radiation, and were allowed to stabilise for 3 minutes before observations were made.

The pulse oximeters were on three trolleys (Table 1). Three groups of 40 patients were studied, and each patient was tested by every pulse oximeter on a given trolley. Each pulse oximeter was tested with two probes, so a second set of pulse oximeter readings with the alternative probe was made on a second run and a second blood sample taken for cooximetry. Thus, a unit that used a finger probe in the first run would then be tested using its ear, nose or fore-

head probe on the second run, or *vice versa*. The three groups of pulse oximeters were tested sequentially in the order given.

#### *Cooximetry and arterial blood gas analysis*

Arterial blood samples were collected anaerobically and stored on ice. Analysis by a cooximeter (IL 482, Instrumentation Laboratory, Lexington, Massachusetts, USA) was performed within one hour of collection. The cooximeter was calibrated each week against the manufacturer's 'Cal Dye' solutions of known haemoglobin concentration. Three test solutions of preserved blood of known saturation supplied by Instrumentation Laboratories were analysed daily to confirm that the performance of the machine was within specification. All procedures were performed according to the manufacturer's instructions.

#### *Statistical analysis*

Descriptive statistics were calculated for all pulse oximeters studied and for the three groups of 40 patients tested using the three groups of pulse oximeters. Comparisons between patient groups were made using a commercial statistics package (Statgraphics, Statistical Graphics Corporation, Rockville, Maryland, USA) and analysis of variance (Scheffe method for parametric data and Kruskal-Wallis method for nonparametric data). The rankings of finger probes and probes in other sites were compared using the Wilcoxon signed rank test for nonparametric data. Statistical significance was accepted when  $p \leq 0.05$ .

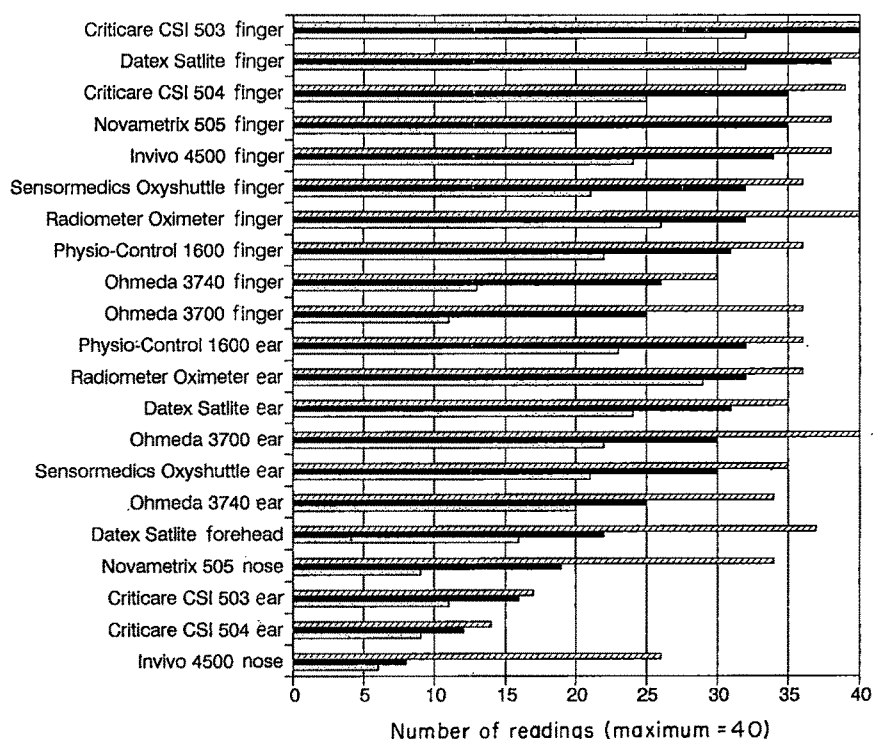
### **Results**

One hundred and twenty patients were studied. The means and standard deviations of all the patient measurements

**Table 1.** Pulse oximeters studied and probes used.

Pulse oximeter	Manufacturer	Probe type
<i>Trolley 1</i>		
Invivo 4500	Invivo Research Labs, Broken Arrow, Oklahoma, USA	Finger, nose
Radiometer	Radiometer A/S, Copenhagen, Denmark	Finger, ear
Ohmeda Biox 3740	Ohmeda, Boulder, Colorado, USA	Finger, ear
Novametrix 505	Novametrix Med. Systems, Wallingford, Connecticut, USA	Finger, nose
Datex	Datex Instrumentarium Corp., Helsinki, Finland	Finger, ear
<i>Trolley 2</i>		
Ohmeda 3700	Ohmeda, Boulder, Colorado, USA	Finger, ear
Physio-Control 1600	Physio-Control, Redmond, Washington, USA	Finger, ear
Sensormedics Oxyshuttle	Sensormedics Corp., Anaheim, California, USA	Finger, ear
Datex	Datex Instrumentarium Corp. Helsinki, Finland	Finger, forehead
<i>Trolley 3</i>		
Criticare CSI 503	Criticare Systems Inc., Milwaukee, Wisconsin, USA	Finger, ear
Criticare CSI 504	Criticare Systems Inc., Milwaukee, Wisconsin, USA	Finger, ear

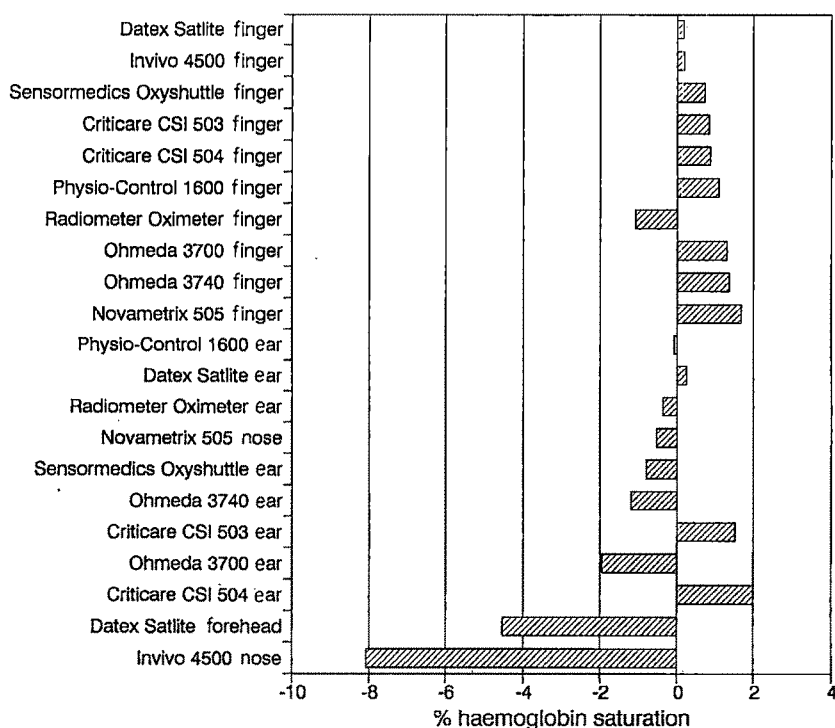




**Fig. 1.** Pulse oximeter finger probes, then probes in other sites, ranked for the total number of readings and those within 3% and 2% of the cooximeter reading. Each pulse oximeter was tested on 40 patients. ▨, total; ■, within 3%; ▩, within 2%.

were reported previously.<sup>7</sup> Analysis of variance showed no difference between the groups with respect to age, rectal temperature, heart rate, cooximeter oxygen saturation, carboxyhaemoglobin, methaemoglobin and blood gas machine bicarbonate and oxygen saturation. Differences

between patients with respect to systolic and diastolic arterial pressures were statistically significant at the 5% level. However, differences in pulse pressures between the three groups were not statistically significant. Given that the mean, systolic and diastolic blood pressures were all



**Fig. 2.** Pulse oximeter finger probes, then probes in other sites, ranked for accuracy, or mean of the difference between the pulse oximeter readings and the cooximeter readings.

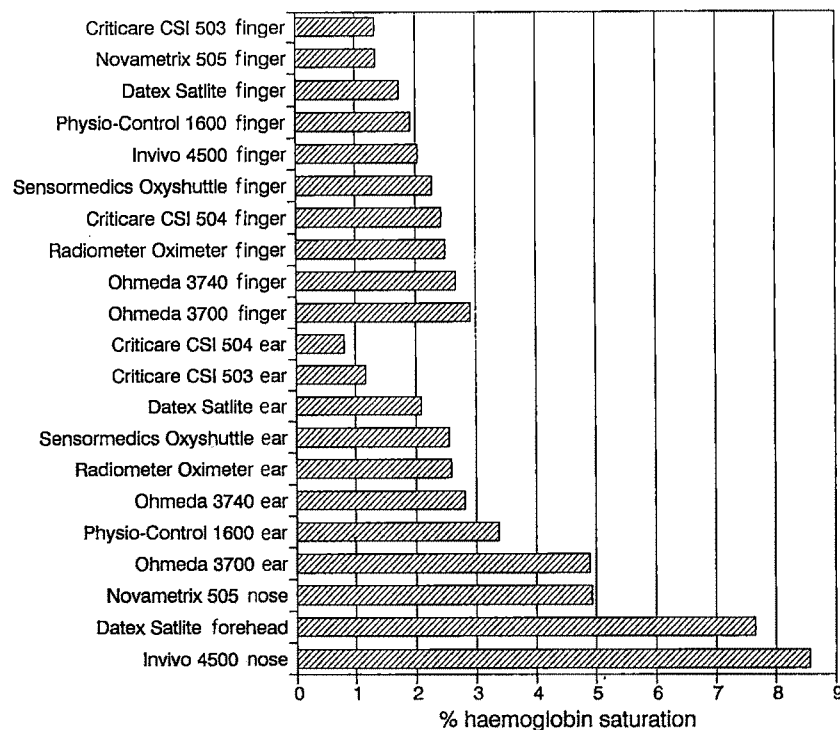


Fig. 3. Pulse oximeter finger probes, then probes in other sites, ranked by precision, or standard deviation of the differences between the pulse oximeter readings and the cooximeter readings.

within the normal clinical range, and there was no difference in pulse pressure, no bias toward the pulse oximeters on any particular trolley was caused by the small differences in systolic and diastolic pressures between the trolleys.

Most of the pulse oximeters failed to give a reading on a number of patients due to poor signal quality. The ability to recognise a weak or very noisy waveform which could cause an erroneous saturation reading is a safety factor of all models used in this trial. Instead of showing saturation the pulse oximeter display is blank, or displays a flashing

value or a message indicating poor signal quality. Three of 10 finger probes and one of eight ear probes gave readings on all 40 patients (Fig. 1).

The absolute mean difference from cooximeter values (bias, 'accuracy') of pulse oximeters ranged from 0.2 to 1.7 for finger probes and 0.1 to 8.1 for other probes (Fig. 2).

The standard deviation (precision) of the differences between pulse oximeter and cooximeter varied from 1.3% to 2.9% in the finger probe group and 0.8% to 8.6% amongst the other probes (Fig. 3).

Table 2. Accuracy of pulse oximeters ranked according to percentage of readings within 3% of the cooximeter readings out of the total number of readings.

Pulse oximeter	Total	Within 3%	Percent within 3%/total	Rank
Criticare CSI 503 finger	40	40	100	1
Datex Satlite finger	40	38	95	2
Criticare CSI 503 ear	17	16	94	3
Novamatrix 505 finger	38	35	92	4
Criticare CSI 504 finger	39	35	90	5
Datex Satlite ear	35	31	89	6
Physio-Control 1600 ear	36	32	89	6
Invivo 4500 finger	38	34	89	6
Radiometer oximeter ear	36	32	89	6
Sensormedics Oxyshuttle finger	36	32	89	6
Ohmeda Biox 3740 finger	28	26	87	11
Criticare CSI 504 ear	14	12	86	12
Physio-Control 1600 finger	36	31	86	12
Sensormedics Oxyshuttle ear	35	30	86	12
Radiometer oximeter finger	40	32	80	15
Ohmeda Biox 3700 ear	40	30	75	16
Ohmeda Biox 3740 ear	34	25	74	17
Ohmeda Biox 3700 finger	36	25	69	18
Datex Satlite forehead	37	22	59	19
Novamatrix 505 nose	34	19	56	20
Invivo 4500 nose	26	8	31	21

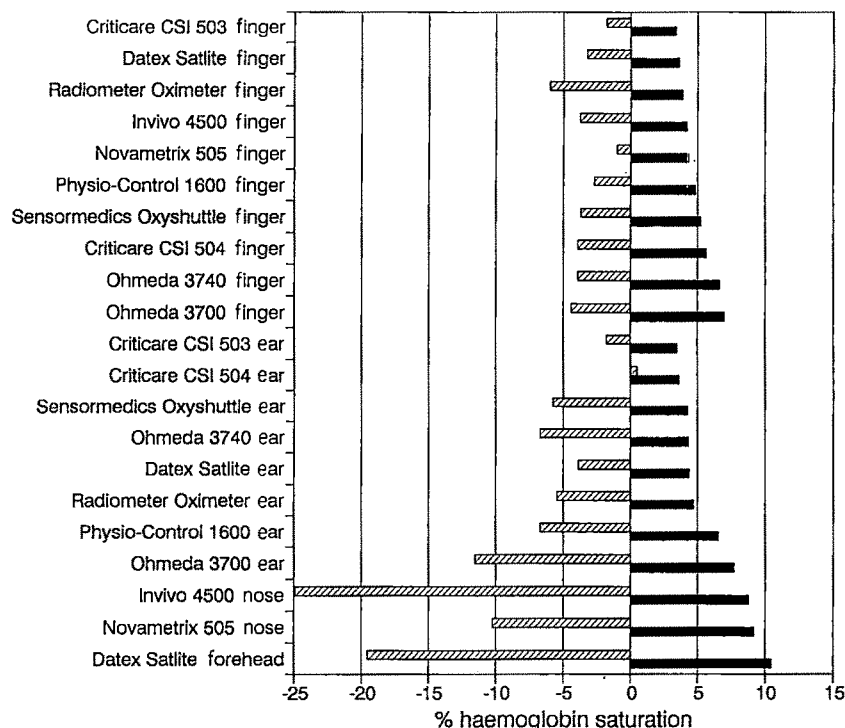


Fig. 4. The 95% limits for each pulse oximeter with finger probes, then probes in other sites, ranked by positive limit.

The number of readings that were within 3% of the cooximeter reading varied from 25 to 40 in the finger probe group compared with 8 to 32 in the alternative probe group (Fig. 1, Table 2).

The percentage of readings within 3% of the cooximeter as a percentage of the total number of readings varied from 69 to 100% in the finger probe group compared with 31 to 94% in the alternative probe group (Table 2).

The fifth method of ranking was to examine the highest positive error (upper 95% confidence interval) below which a reading would be anticipated (Fig. 4). This makes the assumption that, from a clinical point of view, those units which make a lower positive error should be ranked higher than those that make a higher positive error. This varied from 3.42% to 6.98% in the finger probe group compared with 3.57% to 10.47% in the alternative probe group (Fig. 4).

There was no significant difference between the three trolleys with respect to ranking for accuracy, precision, 3% values, percentage of readings within 3%, 95% positive limits or final overall combined ranking using the Kruskal-Wallis test.

The results of average rankings of finger probes and probes in other sites are shown in Table 3. Finger probes

outperformed probes in other sites for each parameter, but this only reached statistical significance for precision and number of readings within 3% of the cooximeter. However, when overall rankings were compared the finger probes were significantly better than probes in other sites.

### Discussion

Manufacturers specify commonly that pulse oximeters have a standard deviation when compared to a 'gold standard', of between 1.5 and 2.5% (more than 75% of units we tested state 2%) at saturations in the range 90–100%. The statistical interpretation of this is that 95% of readings should be within  $1.96 \times$  the standard deviation (4% saturation in the majority) of the true value. Two of 10 finger probes, two of eight ear probes and no nose or forehead probes would be expected to be within 4% of the reference in 95% of readings.

Patients who had undergone cardiopulmonary bypass were chosen for this study because it is documented that they have poor peripheral perfusion,<sup>6,8</sup> and because it is claimed that hypothermia and alterations in peripheral resistance may lead to inaccuracies in pulse oximeter readings.<sup>9</sup> The dropout rate (failure to give any reading) in

Table 3. Comparison of average rankings of finger probes with probes in other sites for accuracy, precision, number of readings within 3%, percentage of readings within 3% out of total number of readings, positive limit and overall ranking (Wilcoxon signed rank test).

	Accuracy	Precision	Number within 3%	Percent within 3%	Positive limit	Overall ranking
Finger	10.0	8.5	6.8	8.7	9.5	8.2
Other site	11.9	13.3	14.9	14.3	12.4	13.5
p	0.50	0.05	0.03	0.09	0.76	0.03

13 units that were used in a previous study of patients after cardiothoracic surgery<sup>7</sup> (53 out of 520 readings) was almost 40 times greater than when the same units were used in our previously reported study on well perfused intensive care patients (3 out of 1141 readings),<sup>5</sup> and thus confirms the relatively poor perfusion status of the patients in this study.

Other workers<sup>4,9-11</sup> have examined how various pulse oximeters perform when peripheral perfusion is reduced, but examined only small numbers of pulse oximeters. Two recent studies have examined how various pulse oximeters respond to rapid decreases in saturation and compared finger and ear probes under these conditions.<sup>12,13</sup> Severinghaus *et al.* produced a rapid plateau of profound hypoxia at 40–70% saturation in volunteers using hypoxic mixtures of oxygen, carbon dioxide and nitrogen. They found that the finger probes responded more slowly than the ear probes and read significantly lower at 55% mean saturation (–6.6(10.8)% for Nellcor N100, and –9.0(10.4)% for the Ohmeda 3700). The mean error with the ear probes was not significant. The 'half-response time' (seconds measured between the computed desaturation transient half-point and the pulse oximeter half-point) T0.5 for ear probes during desaturation was in the range 9.6–19.8 seconds whereas the T0.5 for the finger probes ranged from 24–35.1 seconds. It was suggested that the reason for the slower response time for the finger probes represented the greater transit time for blood to reach the finger compared to the ear, particularly with vasoconstriction in the arm. Kagle *et al.*<sup>13</sup> found that the ear probe using the Ohmeda 3700 pulse oximeter reliably predicted arterial saturation during periods of rapid desaturation, whereas the finger probe significantly underestimated it. The delay time between the ear and finger probe responses was 24 seconds during acute resaturation. Subsequent changes to the algorithm used in the Ohmeda device led to improved agreement with arterial values.

Comparing the eight ear probes that were examined here it is evident that the two Criticare probes had dropout rates greater than 50% but when they did give readings they were the most precise of all the ear probes (Table 2, Figs 1 and 4). They were however among the least accurate (Fig. 2). The Ohmeda 3700 never dropped out, but was the least precise of all the ear probes; it was also one of the least accurate. The Datex, Oxyshuttle and Ohmeda 3700 showed no significant difference between ear and finger probes, the Datex being the most accurate overall.

Nose probes were provided with the Novamatrix and Invivo pulse oximeters. The Invivo in particular had a high dropout rate (Fig. 1), and both units had poor precision (Fig. 3). They appear to have little place in monitoring the patient with poor peripheral perfusion despite their theoretical advantage.

The Datex was the only pulse oximeter that was tested with a forehead reflectance probe. Its accuracy (–4.54%), precision (7.66%) and percentage accuracy (59%) were all very poor despite a low dropout rate. It too seems to have little clinical application.

Comparing the overall performance of the finger probes with probes in other sites we found that the ear, nose and

forehead probes performed worse than the finger probes for all derived parameters (Table 3). No evidence was found to support the use of ear, nose or forehead probes in the poorly perfused patient, unless fingers were unavailable. The faster response time reported by other authors for ear probes is not disputed,<sup>11,12</sup> and the delay with finger probes should be taken into account when planning crisis management algorithms.

Under-reading was a feature of all the probes that performed poorly. This is inherently safer than over-reading, but this cannot be used as a defence for poor performance.

Pulse oximeter users may in clinical practice rotate probes from site to site to obtain the maximum signal response. This strategy, although useful, is not always practical. Where saturation or perfusion may be labile the clinician should choose the pulse oximeter and site most likely to give results of low bias and high precision with a low dropout rate. The results of this study show that in the situation of poor peripheral perfusion forehead and nose probes are unlikely to achieve these goals. Ear probes may be used with some pulse oximeters and results similar in bias and precision to those achieved with finger probes obtained, but often with the risk of a higher dropout rate. We recommend in this clinical situation that, whenever possible, finger probes be used in preference to ear probes.

## References

1. TREMPER KK, BARKER SJ. Pulse oximetry. *Anesthesiology* 1989; **70**: 98–108.
2. ALEXANDER CM, TELLER LE, GROSS JB. Principles of pulse oximetry: Theoretical and practical considerations. *Anesthesia and Analgesia* 1989; **68**: 368–76.
3. KELLEHER JF. Pulse oximetry. *Journal of Clinical Monitoring* 1989; **5**: 37–62.
4. YELDERMAN M, NEW W. Evaluation of pulse oximetry. *Anesthesiology* 1983; **59**: 349–52.
5. EMERGENCY CARE RESEARCH INSTITUTE. Pulse oximeters. *Health Devices* 1989; **18**: 183–230.
6. PALVE H, VUORI A. Pulse oximetry during low cardiac output and hypothermia states immediately after open heart surgery. *Critical Care Medicine* 1989; **17**: 66–9.
7. CLAYTON DG, WEBB RK, RALSTON AC, DUTHIE D, RUNCIMAN WB. A comparison of the performance of twenty pulse oximeters under conditions of poor perfusion. *Anaesthesia* 1991; **46**: 3–10.
8. KUTTLA K, NIINIKOSKI J. Peripheral perfusion after cardiac surgery. *Critical Care Medicine* 1989; **17**: 217–20.
9. TREMPER KK, HUFSTEDLER SM, BARKER SJ, ADAMS AL, WONG DH, ZACCARI J, BENIK K, LEMONS V. Accuracy of a pulse oximeter in the critically ill adult. Effect of temperature and hemodynamics. *Anesthesiology* 1985; **63**: A175.
10. MIHM FG, HALPERIN BD. Noninvasive detection of profound arterial desaturation using a pulse oximetry device. *Anesthesiology* 1985; **62**: 85–7.
11. MORRIS RW, NAIRN M, TORDA TA. A comparison of fifteen pulse oximeters. Part 1: A clinical comparison. Part 2: A test of performance under conditions of poor perfusion. *Anaesthesia and Intensive Care* 1989; **17**: 62–73.
12. SEVERINGHAUS JW, NAIFEH KH. Accuracy of response of six pulse oximeters to profound hypoxia. *Anesthesiology* 1987; **67**: 551–8.
13. KAGLE DM, ALEXANDER CM, BERKO RS, GIUFFRÉ M, GROSS JB. Evaluation of the Ohmeda 3700 pulse oximeter: steady-state and transient response characteristics. *Anesthesiology* 1987; **66**: 376–80.

## The haemodynamic effects of bronchoscopy

### Comparison of propofol and thiopentone with and without alfentanil pretreatment

A. J. HILL, R. O. FENECK, S. M. UNDERWOOD, M. E. DAVIS, A. MARSH AND L. BROMLEY

#### Summary

*The haemodynamic response to bronchoscopy under general anaesthesia was investigated. Forty patients were allocated at random to receive either thiopentone or propofol; half the patients in each group received in addition 18 µg/kg of alfentanil one minute before induction of anaesthesia. The heart rate, noninvasive blood pressure and Holter ECG was monitored in all patients. Significant increases in heart rate ( $p < 0.05$ ), systolic and diastolic arterial pressures ( $p < 0.01$ ) occurred in the thiopentone only group, following bronchoscopy. Systolic and diastolic arterial pressure decreased in patients receiving thiopentone plus alfentanil, following induction of anaesthesia and laryngoscopy ( $p < 0.05$ ). No significant haemodynamic changes were seen in either of the groups which received propofol. ST segment changes on subsequent Holter analysis were seen in four patients, but there were no significant differences between the groups. Anaesthesia with propofol alone provides adequate haemodynamic stability for bronchoscopy and the addition of alfentanil is superfluous.*

#### Key words

*Anaesthetics, intravenous; propofol, thiopentone.  
Analgesics; alfentanil.  
Surgery; bronchoscopy.*

The haemodynamic effects of laryngoscopy and intubation have been described as a sympatho-adrenal response resulting from stimulation of the upper respiratory tract.<sup>1</sup> Increases in heart rate, systemic arterial<sup>2</sup> and pulmonary arterial pressures and right and left heart filling pressures are seen frequently unless active measures are taken to modify this response. The stimulating effects of rigid bronchoscopy, and the resulting haemodynamic responses, have been shown to be qualitatively similar to those of laryngoscopy, but they are often greater and of longer duration.<sup>3</sup> Although these pressor responses are usually well tolerated in the normal circulation, they can have a significant detrimental effect in patients with cardiac disease. In particular, oxygen supply to the heart may be impaired by tachycardia,<sup>4</sup> and myocardial oxygen consumption is increased by hypertension and tachycardia, thereby precipitating episodes of myocardial oxygen imbalance and ischaemia.

Previous work has suggested that pretreatment with a small dose of an opioid may partially obtund the haemodynamic responses to laryngoscopy<sup>5–7</sup> and bronchoscopy,<sup>3</sup> although the effect of opioid pretreatment on the incidence of ischaemic episodes is not so readily demonstrable.<sup>3</sup> Propofol has been shown to be a useful agent in patients

undergoing relatively short procedures,<sup>8</sup> and although the haemodynamic effects of propofol have been likened to those of thiopentone,<sup>9</sup> there is evidence to suggest that the incidence of hypertension and/or tachycardia with propofol anaesthesia would be reduced when compared with thiopentone.<sup>10–12</sup> We have chosen to investigate the haemodynamic responses to bronchoscopy, and compare the effects of propofol with thiopentone anaesthesia, with and without opioid pretreatment with alfentanil.

#### Methods and Materials

Forty patients (ASA 1–3) presenting for elective rigid bronchoscopy under general anaesthesia were included in the trial, which had received the approval of the ethics committee of the National Heart and Chest Hospitals. All the patients were premedicated with intramuscular pethidine 1 mg/kg and atropine 0.4–0.6 mg, given one hour before bronchoscopy. A battery-powered ambulatory electroradiograph (Holter monitor) was attached to the patient using leads in the CM5 and reverse CM5 positions, and this enabled the ECG to be recorded from the time of the premedication until the end of the procedure. On arrival in the anaesthetic room, the patients were connected

A.J. Hill, BSc, MB, ChB, FCAnaes, Research Fellow, R.O. Feneck, MB, BS, FFARCS, Consultant, S.M. Underwood, MB, BS, FFARCS, M.E. Davis, MB, BS, FFARCS, A. Marsh, MB, BS, FFARCS, L. Bromley, MB, BS, FFARCS, Senior Registrars, Department of Anaesthesia, The London Chest Hospital, Bonner Road, London E2 9JX.

Correspondence should be addressed to Dr R.O. Feneck please.

Accepted 16 August 1990.



to a noninvasive blood pressure monitor (Datex Accutor) and the standard lead II of the ECG was monitored and displayed throughout the study. Systemic arterial blood pressure was monitored at one-minute intervals.

The patients were allocated at random to four groups: group P, propofol; group T, thiopentone; group P/A, propofol + alfentanil; group T/A, thiopentone + alfentanil.

In all patients, intravenous access was established, and patients in groups P/A and T/A received alfentanil 18 µg/kg, followed one minute later by propofol or thiopentone. Patients in groups P and T received the induction agent alone; in all patients sufficient propofol or thiopentone was given to obtund the eyelash reflex and muscular relaxation was achieved with suxamethonium 1 mg/kg. The patients' lungs were ventilated with 100% oxygen via a facemask and, when full muscular relaxation had occurred, direct laryngoscopy was performed and the larynx sprayed with 4 ml of 4% lignocaine.

Rigid bronchoscopy was performed with a Negus bronchoscope and ventilation maintained by use of a Sanders injector using 100% oxygen. Further increments of anaesthetic and relaxant were given as required, not less than 20 mg propofol or 50 mg thiopentone every 2 minutes, and not less than 25 mg suxamethonium every 5 minutes. At the end of the study period, a blood sample was taken from the radial artery for blood gas analysis.

Haemodynamic variables were recorded at six points during the study: at baseline i.e. before induction of anaesthesia; following induction; following laryngoscopy and topical lignocaine; following insertion of the rigid bronchoscope; 2 minutes after insertion of the bronchoscope; 5 minutes after insertion of the bronchoscope.

Statistical analysis was carried out using the first five time points only because the procedures varied in duration. Analysis was performed comparing baseline data by Kruskal-Wallis one-way analysis of variance, and between-

group variation through time was investigated using the Friedman two-way analysis of variance by ranks. Within-group analysis was undertaken by Wilcoxon matched pairs, and Student's *t*-test, as appropriate.

## Results

The patient data for each group are shown in Table 1, and as can be seen the groups are comparable on the basis of age and body weight, and on the dose of suxamethonium used both following induction and throughout the procedure. Between-group comparison showed that the addition of alfentanil resulted in a significant reduction in the amount of thiopentone given at induction ( $p < 0.01$ ), but the addition of alfentanil did not result in any reduction in the dose of propofol administered. In both groups, the addition of alfentanil resulted in an overall reduction in the total dose of intravenous agent used during the study, although this difference is only statistically significant between groups P and P/A.

Table 2 shows the changes in heart rate (HR) which occurred in each group during the study period. Analysis shows that there are no significant differences between the groups at baseline. In group T (thiopentone only), there are significant increases in HR during the study period ( $p < 0.05$ ), with the maximum increase occurring at time point 4.

Table 3 shows the data for systolic blood pressure (SAP). There are no significant differences between the groups at baseline. Within-group analysis shows that SAP changes significantly in both groups T and T/A. In group T there are significant increases in SAP during the study which are maximal following bronchoscopy time 4, ( $p < 0.05$ ), whereas in group T/A SAP decreased, maximal at time 3. There were no significant changes in either groups P or P/A.

Table 1. Demographic data; mean (SEM).

Group	P	T	P/A	T/A
Age; years	61 (3.3)	60 (4.7)	58 (3.7)	61 (3.5)
Weight; kg	71.6 (4.2)	70.3 (4.4)	69.2 (3.1)	66.4 (3.9)
Induction agent				
Initial dose; mg	153 (14)	380 (26)*	146 (12)	282 (30)
Total	296 (20)**	555 (42)	214 (18)	458 (35)
Suxamethonium				
Initial dose; mg	72 (4.8)	73 (3.9)	70 (3.9)	69 (3.4)
Total	111 (16)	108 (13)	93 (6)	99 (6)

\*probability  $< 0.01$  group T compared to group TA.

\*\*probability  $< 0.01$  group P compared to group PA.

Table 2. Heart rate, recorded at six points during the study; beats per minute, mean (SEM).

Group	One	Two	Three	Four	Five	Six
P	89 (7)	85 (7)	91 (5)	93 (5)	90 (6)	88 (8)
T	91 (4)	91 (4)	93 (5)	97 (6)*	95 (6)	89 (6)
P/A	93 (6)	97 (5)	94 (4)	91 (4)	94 (4)	88 (4)
T/A	83 (4)	81 (5)	80 (3)	84 (6)	83 (5)	89 (4)

\*probability  $< 0.05$  compared to baseline.

**Table 3.** Systolic arterial pressure, recorded at six points during the study; mmHg, mean (SEM).

Group	One	Two	Three	Four	Five	Six
P	139 (7)	120 (8)	132 (10)	135 (9)	130 (6)	119 (15)
T	131 (6)	116 (4)	136 (9)	155 (8)*	143 (9)	120 (10)
P/A	126 (6)	105 (2)	110 (5)	116 (6)	106 (6)	106 (8)
T/A	120 (4)	101 (5)***	97 (5)***	116 (6)	106 (3)	106 (7)

\*probability &lt; 0.05 compared to baseline.

\*\*\*probability &lt; 0.001 compared to baseline.

**Table 4.** Diastolic arterial pressure, recorded at six points during the study; mmHg, mean (SEM).

Group	One	Two	Three	Four	Five	Six
P	83 (6)	75 (4)	86 (7)	87 (7)	78 (4)	79 (11)
T	85 (5)	77 (2)	90 (5)	97 (6)*	89 (5)	82 (7)
P/A	72 (4)	58 (2)	59 (5)	65 (4)	60 (6)	59 (5)
T/A	73 (2)	58 (5)**	54 (4)**	69 (5)	75 (3)	70 (7)

\*probability &lt; 0.025 compared to baseline.

\*\*probability &lt; 0.01 compared to baseline.

Table 4 shows the data for diastolic blood pressure (DAP), which mirror those changes seen with the SAP in that group T shows a significant increase in DAP ( $p < 0.025$ ), group T/A shows a significant reduction in DAP ( $p < 0.01$ ), and no significant changes are seen in groups P and P/A.

Table 5 shows the data obtained from blood gas and ST segment analysis. As can be seen the groups were normocapnic and well oxygenated, and there were no significant differences between them. The distribution of ST segment change between groups is not significant. There were four patients who had ST segment changes of greater than 0.1 mV for longer than 60 seconds, compatible with criteria for diagnosis of developing myocardial ischaemia. This represents an incidence of 10%.

Table 6 demonstrates the range of haemodynamic change, blood gas analysis and magnitude of deviation that

occurred in patients who developed ST segment abnormality.

### Discussion

Previous work has suggested that rigid bronchoscopy following intravenous anaesthesia is associated with a significant haemodynamic response which is not controlled by either the anaesthetic premedication or topical laryngeal lignocaine, and which is only partially controlled by the addition of a small dose of an opioid.<sup>3</sup> These observations which refer only to intravenous anaesthesia with thiopentone, are in agreement with the findings of this study.

Patients who received only thiopentone had an increase in HR, SAP and DAP, whereas in those patients who received alfentanil followed by thiopentone the SAP and DAP decreased. In contrast, in the patients who received

**Table 5.** Arterial blood gases and ST segment; mean (range).

Group	P	T	P/A	T/A
Saturation; %	99.4	99.4	99.4	97.9
$P_{aO_2}$ ; kPa	29.9	29.7	28.4	27.7
	(15.3–55.6)	(15.6–52.9)	(19.1–55.5)	(12.9–48.7)
$P_{aCO_2}$ ; kPa	5.95	5.49	5.71	5.6
	(4.79–7.27)	(3.57–7.11)	(4.23–6.59)	(3.52–6.95)
ST segment; number	1	0	1	2

**Table 6.** Details of patients with ST segment changes (range).

Patients	Heart rate	SAP mmHg	DAP mmHg	$P_{aO_2}$ kPa	$P_{aCO_2}$ kPa	ST mm
	beats/minute					
P (1)	78–116	89–151	66–88	30.9	7.27	–1.0
P/A (2)	66–82	85–130	38–72	55.5	5.36	–1.0
T/A (6)	88–116	105–143	59–77	30.0	3.52	+2.0
T/A (8)	65–104	94–138	54–76	48.7	5.45	+2.5

propofol, with or without alfentanil, no significant changes in HR, SAP or DAP were seen.

Previous work has suggested that induction of anaesthesia with propofol is associated with an equal<sup>9</sup> or greater<sup>10-12</sup> degree of hypotension when compared with thiopentone, both in normals and in patients with ischaemic heart disease.<sup>9,13</sup> In this study, induction with propofol was associated with an equal reduction in blood pressure compared with thiopentone, but there was no significant increase in HR. This absence of tachycardia with propofol has been described before,<sup>10</sup> although the exact mechanism remains unclear. However, the hypertension and tachycardia seen with thiopentone during the study period, appears to be well controlled in propofol anaesthesia, and no significant changes are seen in groups P and P/A throughout the study period. All the patients in this study had comparable premedication, and received similar doses of suxamethonium during the study. Furthermore, haemodynamic differences cannot be attributed to hypoxia or hypercarbia, since these patients were well oxygenated and normocarbic on blood gas analysis (Table 5). Although we have used a physiological endpoint to control the induction dose of thiopentone or propofol, it is interesting to note that the calculated potency ratio between the two induction agents used in the study is almost exactly that reported elsewhere, i.e. 2.5 : 1.0. Thus the haemodynamic differences seen cannot be explained on the basis of the administration of non-equipotent doses of induction agent. We believe the differences between thiopentone and propofol observed in this study to be real, and suggest that intravenous anaesthesia for rigid bronchoscopy is associated with greater haemodynamic stability when propofol is used than with thiopentone. Furthermore, no benefit could be attributed to the addition of alfentanil to propofol, in contrast to thiopentone.

There are few data available on the incidence of ischaemia occurring during the course of rigid bronchoscopy. Our previous study, which also used Holter ECG monitoring, suggested that 18% (6/36) of patients developed ischaemic ST segment changes during the study.<sup>3</sup> On this occasion 10% (4/40) of patients demonstrated ST segment changes. These findings reflect a high incidence of ischaemia, particularly since these patients were asymptomatic for ischaemic heart disease, although by nature of their age and pulmonary disease, it should be said that they have risk factors in common. Nevertheless, it is important to establish how likely it is that these ST segment changes are true indicators of developing ischaemia.

Recent evidence has suggested that unipolar standard lead ECG monitoring is not effective in detecting the majority of ischaemic episodes that occur in the peri-operative period.<sup>14-16</sup> Three- and four-lead monitoring systems have been suggested as a suitable alternative.<sup>16</sup> The Holter monitor has also been recommended for use in this period<sup>17,18</sup> and it has been used extensively in the investigation of heart disease, both to detect ischaemic episodes<sup>19,20</sup> and arrhythmias.<sup>21</sup> The use of the Holter monitor with the CM5 and reverse CM5 electrode positions allowed us to detect anterior, lateral and posterior changes, and continuous monitoring of standard lead II allowed detection of inferior ischaemic changes. The degree and duration of any ST segment changes must be recorded if their significance is to be determined. In this situation, it is necessary to strike a

correct balance between sensitivity and specificity for the diagnosis of ischaemic change. Thus we have chosen to take a change of 0.1 mV for a minimum duration of 60 seconds, and to disregard any ST segment changes not fulfilling these criteria. All ST segment changes to be identified as ischaemic in origin should have a specific profile in relation to the J point.

In order to confirm the aetiology of supposedly significant ST segment change, it is advisable to consider other factors. Thus although false-positives do occur, these are more likely in low risk groups (i.e. young, females, non-smokers) than in high risk groups (i.e. elderly, male, smokers). In addition, a positive family history may be especially relevant in young asymptomatic patients. It can be seen from Table 6 that none of the patients who developed significant ST segment changes had any gross haemodynamic disturbance associated with the ischaemia. Analysis of the blood gases also failed to demonstrate any significant hypoxia or hypercarbia. Frequently, haemodynamic changes, in particular tachycardia, may be seen, but ST segment abnormality should certainly not be dismissed as artifact in the absence of gross haemodynamic disorder. Previous work has established that peri-operative ischaemia in asymptomatic patients with proven coronary artery disease may occur without the development of significant haemodynamic abnormality.<sup>22</sup>

Although ST segment monitoring commenced following premedication, all the significant changes seen in this study occurred during the period of anaesthesia and bronchoscopy. Review of the case histories and of the strength of the ST segment changes seen, leads us to conclude that all the episodes are true positives. Thus both this and our previous study shows that there is an unacceptably high incidence of peri-operative ischaemia in patients undergoing pulmonary surgery. Many would doubt that rigid bronchoscopy even represents the time of maximum risk of ischaemia during the procedure. In particular one-lung ventilation would appear to be a period of greater potential stress, especially if it is associated with severe arterial desaturation.

If the incidence of peri-operative ischaemia during rigid bronchoscopy is indeed 10–15% as our studies suggest, then it may be that the incidence of peri-operative ischaemia during pulmonary resection is much higher. There do not appear to be any data to support or refute this supposition, but if true it may help to explain why peri-operative mortality following pneumonectomy or lobectomy is approximately twice the peri-operative mortality following coronary artery surgery, both at this institution and nationally.<sup>23</sup> We believe that a study designed to record the incidence of peri-operative ischaemia in patients undergoing pulmonary surgery is therefore urgently needed, in order to establish both the nature and the scale of the problem.

## References

1. TOMORI Z, WIDDICOMBE JG. Muscular, bronchomotor and cardiovascular reflexes elicited by mechanical stimulation of the respiratory tract. *Journal of Physiology* 1969; **200**: 25–49.
2. MILLAR FORBES A, DALLY FG. Acute hypertension during induction of anaesthesia and endotracheal intubation in normotensive man. *British Journal of Anaesthesia* 1970; **42**: 618–25.
3. WARK KJ, LYONS J, FENECK RO. The haemodynamic effects of

- bronchoscopy. Effect of pretreatment with fentanyl and alfentanil. *Anaesthesia* 1986; **41**: 162-7.
4. SLOGOFF S, KEATS AS. Does perioperative myocardial ischemia lead to postoperative myocardial infarction? *Anesthesiology* 1985; **62**: 107-14.
5. DAHLGREN N, MASSETER K. Treatment of stress response to laryngoscopy and intubation with fentanyl. *Anaesthesia* 1981; **36**: 1022-66.
6. KAUTTO UM. Attenuation of the circulatory response to laryngoscopy and intubation by fentanyl. *Acta anaesthesiologica Scandinavica* 1982; **26**: 217-21.
7. HARRIS CE, MURRAY AM, ANDERSON JM, GROUNDS RM, MORGAN M. Effects of thiopentone, etomidate and propofol on the haemodynamic response to tracheal intubation. *Anaesthesia* 1988; **43** (Suppl.): 32-6.
8. NOBLE J, OGG TW. The effect of propofol ('Diprivan') and methohexitone on memory after day case anaesthesia. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 103-4.
9. STEIB A, FREYS G, BELLER JP, CURZOLA U, OTTENI JC. Propofol in elderly high risk patients. A comparison of haemodynamic effects with thiopentone during induction of anaesthesia. *Anaesthesia* 1988; **43** (Suppl.): 111-14.
10. GROUNDS RM, TWIGLEY AJ, CARLI F, WHITWAM JG, MORGAN M. The haemodynamics effects of intravenous induction. Comparison of the effects of thiopentone and propofol. *Anaesthesia* 1985; **40**: 735-40.
11. MCCOLLUM JSC, DUNDEE JW. Comparison of induction characteristics of four intravenous anaesthetic agents. *Anaesthesia* 1986; **41**: 995-1000.
12. FAHY LT, VAN MOURIK GA, UTTING JE. A comparison of the induction characteristics of thiopentone and propofol (2, 6-diisopropyl phenol) *Anaesthesia* 1985; **40**: 939-44.
13. PATRICK MR, BLAIR IJ, FENECK RO, SEBEL PS. A comparison of the haemodynamic effects of propofol ('Diprivan') and thiopentone in patients with coronary artery disease. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 23-7.
14. GRIFFIN RM, KAPLAN JA. Myocardial ischaemia during non-cardiac surgery. *Anaesthesia* 1987; **42**: 155-9.
15. ROY WL, EDELST G, GILBERT B. Myocardial ischemia during non-cardiac surgical procedures in patients with coronary-artery disease. *Anesthesiology* 1979; **51**: 393-7.
16. LONDON MJ, HOLLENBERG M, WONG MG, LEVENSON L, TUBAU JF, BROWNER W, MANGANO DT AND THE SPI RESEARCH GROUP. Intraoperative myocardial ischemia: localization by continuous 12-lead electrocardiography. *Anesthesiology* 1988; **69**: 232-41.
17. KNIGHT AA, HOLLENBERG M, LONDON MJ, TUBAU J, VERRIER MD, BROWNER W, MANGANO DT AND THE SPI RESEARCH GROUP. Perioperative myocardial ischemia: importance of the preoperative ischemic pattern. *Anesthesiology* 1988; **68**: 681-8.
18. DODDS TM, DELPHIN E, STONE JG, GRAL SG, COROMILAS J. Detection of perioperative myocardial ischemia using Holter monitoring with real-time ST segment analysis. *Anesthesia and Analgesia* 1988; **67**: 890-3.
19. ROZANSKI A, BERMAN DS. Silent myocardial ischaemia. 1. Pathophysiology, frequency of occurrence, and approaches toward detection. *American Heart Journal* 1987; **114**: 615-26.
20. DEANFIELD JE, RIBIERO P, OAKLEY K, KRIKLER S, SELWYN AP. Analysis of ST-segment changes in normal subjects: implications for ambulatory monitoring in angina pectoris. *American Journal of Cardiology* 1984; **54**: 1321-1325.
21. PRATT CM, SLYMEN DJ, WIERMAN AM, YOUNG JB, FRANCIS MJ, SEALS AA, QUINONES MA, ROBERTS R. Analysis of the spontaneous variability of ventricular arrhythmias: consecutive ambulatory electrocardiographic recordings of ventricular tachycardia. *American Journal of Cardiology* 1985; **56**: 67-72.
22. CAMPBELL S, BARRY J, REBECCA GS, ROCCO MB, NABLE EG, WAYNE RR, SELWYN AP. Active transient myocardial ischemia during daily life in asymptomatic patients with positive exercise tests and coronary artery disease. *American Journal of Cardiology* 1986; **57**: 1010-16.
23. UNITED KINGDOM THORACIC SURGICAL REGISTER. 1985-6-7. Combined mortality for lobectomy and pneumonectomy.

## The pharmacodynamics of alcuronium in the elderly

A. P. KENT AND J. M. HUNTER

### Summary

*Alcuronium (0.2 mg/kg) was given to 12 elderly patients, mean age 77 years (range 70–88 years) and 12 young patients, mean age 24 years (range 18–32 years) undergoing general anaesthesia. A compound muscle action potential was monitored continuously throughout anaesthesia, using an electromyograph and the train-of-four twitch technique. The rate of onset and maximum block achieved were similar in both the young and elderly patients, as were the times to 20% recovery of the first twitch compared with control (T1:T0) and fourth twitch compared with the first, (T4:T1). In contrast, the time to 70% recovery of T1:T0 was significantly prolonged in the elderly (138 as compared with 89 minutes;  $p < 0.01$ ) as was the recovery index (25–75%) for T1:T0 (95 as compared with 46 minutes;  $p < 0.01$ ) and the time to 70% recovery of T4:T1 (181 as compared with 131 minutes;  $p < 0.05$ ). The recovery curves for T1:T0 and T4:T1 were also significantly different in the elderly from the young group ( $p < 0.01$  in both instances). These results show that the duration of action of alcuronium is significantly prolonged in the elderly.*

### Key words

Neuromuscular relaxants; alcuronium.

Pharmacodynamics.

Age factors; geriatric.

Alcuronium is an established nondepolarising neuromuscular blocking agent with a similar duration of action to tubocurarine,<sup>1</sup> which has been widely used in all age groups since 1961. It is thought to be partly metabolised by the liver but also excreted through the kidney.<sup>2</sup> The function of both these organs is known to deteriorate with increasing age,<sup>3</sup> so the duration of action of alcuronium may be prolonged in the elderly. However, although the effect of advancing age has been shown to prolong the action of many nondepolarising muscle relaxants including tubocurarine,<sup>4</sup> pancuronium<sup>5,6</sup> and possibly vecuronium,<sup>6</sup> there is little information available on the use of alcuronium in the elderly. A purely pharmacokinetic study of alcuronium by Stephens and colleagues suggested that the clearance was reduced and the elimination half-life prolonged in the aged, but their results were complicated by the fact that the relationship was not substantiated in a subgroup of elderly patients who had experienced significant intra-operative blood loss.<sup>7</sup>

This present study attempts a comparison of the pharmacodynamics of alcuronium in elderly patients with a group of healthy young adults.

### Methods

Twelve elderly patients, mean age 78 years (range 70–88 years) and 12 young patients, mean age 24 years (range 18–32 years) were studied. They were presenting for elective surgery, during which minimal blood loss was anticipated and in which tracheal intubation was an accepted part of the anaesthetic technique (e.g. laparoscopy or closure of colostomy). All gave informed consent and the study was approved by the Hospital Ethics Committee. Both groups consisted of six male and six female patients: the mean weight in the elderly group was 62 kg (range 50–84 kg) and in the young group, 69 kg (range 45–98 kg). Only patients of ASA groups 1 or 2 were included and all patients studied were shown to have normal renal and hepatic function on routine pre-operative screening.

The patients were premedicated with intramuscular morphine 5–10 mg and cyclizine 25–50 mg, 1 hour before induction of anaesthesia with thiopentone 4–5 mg/kg in combination with fentanyl 50–100 µg and midazolam 2–5 mg. Anaesthesia was maintained with 70% nitrous oxide in oxygen, while control values of the compound muscle

A.P. Kent, MB, ChB, FFARCS, Lecturer, J.M. Hunter, MB, ChB, FFARCS, Senior Lecturer, University Department of Anaesthesia, Royal Liverpool Hospital, Prescot Street, P.O. Box 147, Liverpool L69 3BX.

Accepted 11 June 1990.



**Table 1.** The times to, and degree of, maximum depression of T1:T0 and T4:T1 in the young and elderly groups expressed as a mean, (standard deviation) and [range].

	Young (n = 12)	Elderly (n = 12)	
Time to maximum depression T1:T0; minutes	7.1 (3.2) [3.7–14.0]	7.0 (4.5) [2.3–16.0]	ns
Time to maximum depression T4:T1; minutes	25 (18.4) [4.8–59.0]	25.3 (24.5) [4.0–68.0]	ns
Maximum depression T1:T0; %	93.5 (5.4) [86–99]	93.9 (5.6) [79–99]	ns
Maximum depression T4:T1; %	88.1 (12.8) [66–100]	92.2 [10.5] [66–100]	ns

ns, not significant

action potential (CMAP) from the *adductor pollicis* were obtained by stimulating the ulnar nerve in the forearm using the train-of-four twitch technique (To4) with a Medelec MS6 electromyograph.<sup>8</sup> After stable control values of the To4 had been obtained, alcuronium 0.2 mg/kg was given into an intravenous infusion in the opposite arm. Tracheal intubation was performed when clinically appropriate and anaesthesia maintained throughout surgery using nitrous oxide 70% in oxygen with increments of fentanyl and midazolam as indicated. No other volatile agent was added and no further alcuronium administered. The To4 was monitored throughout anaesthesia at 12.5-second intervals.

No neostigmine was given at the end of surgery and anaesthesia was continued until the height of the first twitch of the ToF (T1) had reached at least 75% of control (T0) and the height of the fourth twitch (T4) had reached at least 70% of T1. Anaesthesia was then discontinued and the patients extubated before transfer to the recovery room where satisfactory recovery of muscle tone was checked by asking the patient to cough, lift their head off the pillow for 5 seconds and protrude the tongue.<sup>8</sup> Each patient was also interviewed 24 hours later to seek any evidence of dreaming or awareness.

From the electromyograph recording, the heights of T1:T0 and T4:T1 were calculated at one-minute intervals after the bolus of alcuronium. The unpaired Wilcoxon rank test was used to compare the two groups with respect to the time to, and the degree of, maximum depression of T1:T0 and T4:T1, the time from injection of relaxant to 20% and 70% recovery of T1:T0 and T4:T1, and the recovery index for T1:T0 (25–75%). The recovery curves of T1:T0 and T4:T1 in the young and elderly were also compared using an analysis of variance.

### Results

The onset data are shown in Table 1. There was no significant difference between the young and elderly groups in the times to maximal depression of T1:T0 or T4:T1, or in the degree of maximal block.

The recovery data are shown in Table 2. The times to 20% recovery of T1:T0 and T4:T1 were not significantly different between the two groups. The times to 70% recovery of T1:T0 and T4:T1 were, however, significantly longer in the elderly group ( $p < 0.01$  and  $p < 0.05$  respectively). A comparison of the recovery index (25–75%) for

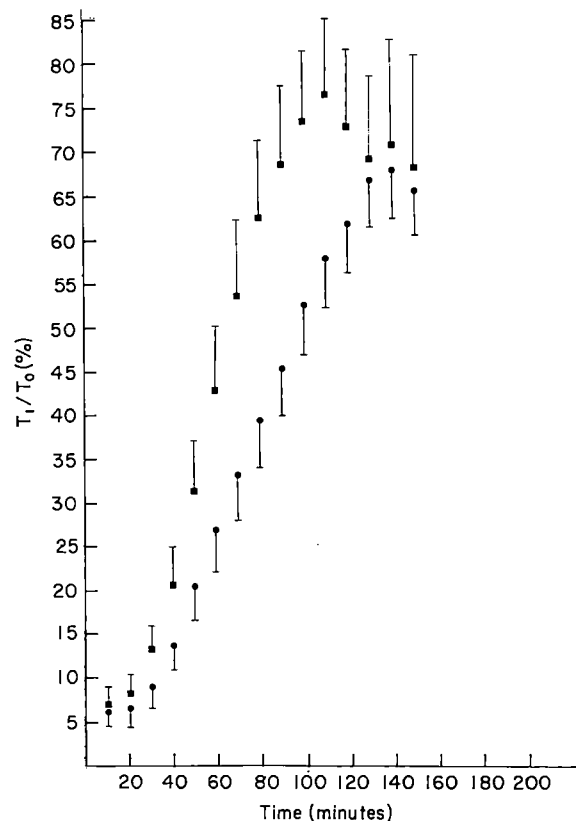
**Table 2.** The recovery data for T1:T0 and T4:T1 are given in the young and elderly groups. Time in minutes is expressed as a mean, (standard deviation) and [range]. There is a significant difference between the two groups using the unpaired Wilcoxon rank test \* =  $p < 0.05$ ; \*\* =  $p < 0.01$ .

	Young (n = 12)	Elderly (n = 12)	
T1:T0			
20% Recovery; minutes	47.2 (23.8) [19–90]	54.5 (23.6) [6–94]	ns
70% Recovery; minutes	89.9 (41.1) [54–192]	138.8 (47.3) [91–233]	**
Recovery index; minutes (25–75%)	46.2 (27.5) [23–126]	95.3 (45) [64–183]	**
T4:T1			
20% Recovery; minutes	77.8 (13.1) [58–97]	77.3 (23.3) [51–124]	ns
70% Recovery; minutes	131.3 (35.7) [100–211]	181.8 (59.7) [113–307]	*

ns, not significant

T1:T0 also revealed a significant prolongation in the elderly group ( $p < 0.01$ ).

The recovery profiles of T1:T0 in the young and elderly groups are shown in Figure 1. An analysis of variance showed a significant difference ( $p < 0.01$ ) between the recovery curves of the young and elderly patients. Similarly, the recovery profile of T4:T1 in the two groups

**Fig. 1.** The recovery of T1:T0 (%) as compared with time (minutes) expressed as a mean with bars indicating SEM, is plotted for the young (■) and elderly (●) patients. The mean values for the last three time points in the young group are respectively for six, five and four patients only.

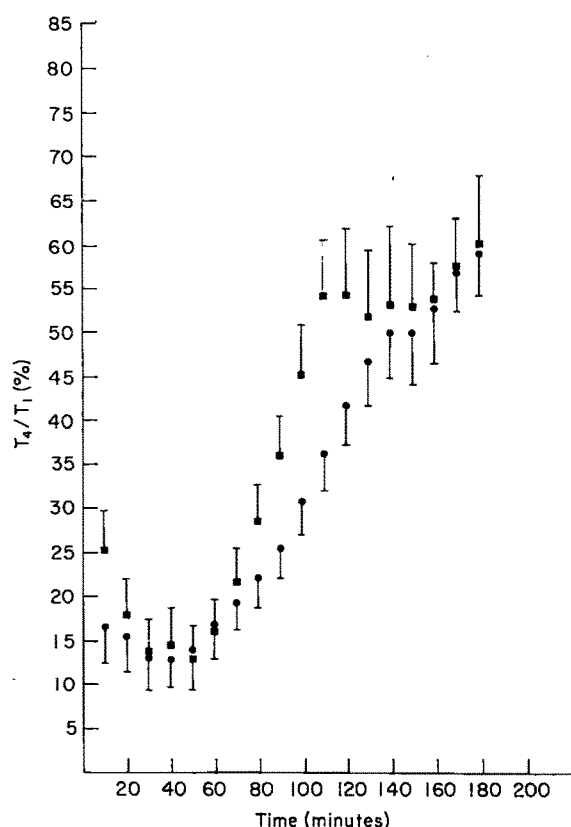


Fig. 2. The recovery of T4:T1 (%) as compared with time (minutes) expressed as a mean with bars indicating SEM, is plotted for the young (■) and elderly (●) patients. The mean values for the last three time points in the young group are for respectively four, three and three patients only

is shown in Figure 2. An analysis of variance again showed a significant difference ( $p < 0.01$ ) between the young and elderly groups.

When interviewed 24 hours after surgery none of the patients reported any episode of dreaming or awareness during anaesthesia. Similarly, no symptoms suggestive of residual curarisation, e.g. double vision, were reported.

### Discussion

The prolongation of action of many neuromuscular blocking agents in the elderly, including tubocurarine,<sup>4</sup> pancuronium<sup>5</sup> and possibly vecuronium,<sup>6</sup> has been considered to be due to the gradual deterioration in renal and hepatic function with increasing age.<sup>9</sup> In the early stages this deterioration may not be detected by routine laboratory screening. Indeed, an increased elimination half-life has been demonstrated for atracurium in this age group, which suggests that atracurium clearance is, at least to a small extent, also dependent on organ function.<sup>10</sup> Little information is available on the action of alcuronium in the aged. A single pharmacokinetic study<sup>7</sup> showed a delayed clearance and increased elimination half-life in five elderly patients undergoing minor surgery when compared with values for these variables reported in young healthy adults by Walker and colleagues,<sup>2</sup> but the results were confused by the failure to confirm them in elderly patients having major surgical procedures.<sup>7</sup> The current pharmacodynamic study

confirms these findings, and demonstrates that the recovery of the To4 twitch response, both in terms of T1:T0 and T4:T1, is significantly delayed in old age.

Previous workers have expressed concern that when the EMG is used, recovery from neuromuscular blockade may appear incomplete because of a change in the latency and duration of the electrical response.<sup>11</sup> Both groups of patients in this study received the same muscle relaxant and underwent identical monitoring for similar periods of time, so any such change should be similar in both groups and therefore of limited significance. In our investigation, which was performed on patients undergoing minor surgery of short duration, we did not have the opportunity to determine whether return to control values always occurred, although 11 out of 12 young, and seven out of 12 elderly patients achieved at least 80% return of T1:T0. The data displayed on the recovery curves for both T1:T0 and T4:T1 in the elderly patients (Figs 1 and 2) are therefore more limited than was first intended since further data collection would have prolonged the anaesthetic time far beyond that required for surgery. Despite this limitation, the clinical recovery of the elderly patients was, without exception, very satisfactory. Such limited recovery data are not seen in the young patients; the reduced number of young patients at the later recorded times is because many patients had already fully recovered from neuromuscular block.

In contrast, the onset of neuromuscular block in this study, in terms of degree and time to maximum block after the bolus dose, was not significantly different in either group. Donati has reviewed factors affecting onset of action of muscle relaxants and noted the particular importance of cardiac output in this respect.<sup>12</sup> The similar onset data found in this study are probably a reflection of the good myocardial status of the elderly patients; this similarity may not have been demonstrated if the older patients had not been fit. This finding is not new; previous dose-response studies performed in the elderly for tubocurarine by Matteo<sup>4</sup> and more recently for pancuronium, vecuronium and atracurium by Bell and colleagues<sup>13</sup> have failed to show a difference in the degree of maximum block and in the time for this to be achieved.

The mean time to onset of maximum neuromuscular block, over 7 minutes in both groups of patients, is perhaps longer than would be anticipated. Although it is realised that only a relatively small dose of alcuronium was used in this study (0.2 mg/kg), the long onset of action with a wide range of results is similar to that found with tubocurarine and pancuronium<sup>14</sup> and must be considered when alcuronium is being used before tracheal intubation. Also of note are the mean times to 20% recovery of T1:T0; 47 minutes even in the young patients, which would suggest that alcuronium should not be considered a muscle relaxant of only intermediate duration.

No potent inhalational agent was used in this study as halothane, enflurane and isoflurane have all been shown markedly to potentiate the neuromuscular block produced by alcuronium.<sup>15</sup> The degree of this potentiation is variable and to have included an inhalational agent in our anaesthetic technique would probably have increased the range of recovery times. The hypnotic midazolam was therefore used in combination with nitrous oxide and fentanyl. Although midazolam has been shown to potentiate the neuromuscular block produced by vecuronium and tubo-

curarine in animals, the effect is only produced at much larger doses than those used in this study.<sup>16</sup>

#### Acknowledgments

We are grateful to Mr J. D. Taylor for his patience and cooperation during this study. The electromyograph was bought with a grant from the Mersey Regional Health Authority.

#### References

1. LUND I, STOVNER J. Experimental and clinical experiences with a new muscle relaxant Ro 4-3816, diallyl-nor-toxiferine. *Acta Anaesthesiologica Scandinavica* 1962; **6**: 85-97.
2. WALKER J, SHANKS CA, TRIGGS EJ. Clinical pharmacokinetics of alcuronium chloride in man. *European Journal of Clinical Pharmacology* 1980; **17**: 449-57.
3. MILLAR R. Anesthesia for the aged: some pharmacokinetic and pharmacodynamic considerations. *The Mount Sinai Journal of Medicine* 1987; **54**: 305-7.
4. MATTEO RS, BACKUS WW, McDANIEL DD, BROTHERTON WP, ABRAHAM R, DIAZ J. Pharmacokinetics and pharmacodynamics of d-tubocurarine and metocurine in the elderly. *Anesthesia and Analgesia* 1985; **64**: 23-9.
5. DUVALDESTIN P, SAADA J, BERGER JL, D'HOLLANDER A, DESMONTS JM. Pharmacokinetics, pharmacodynamics, and dose-response relationships of pancuronium in control and elderly subjects. *Anesthesiology* 1982; **56**: 36-40.
6. RUPP SM, CASTAGNOLI KP, FISHER DM, MILLER RD. Pancuronium and vecuronium pharmacokinetics and pharmacodynamics in younger and elderly adults. *Anesthesiology* 1987; **67**: 45-9.
7. STEPHENS ID, HO PC, HOLLOWAY AM, BOURNE DWA, TRIGGS EJ. Pharmacokinetics of alcuronium in elderly patients undergoing total hip replacement or aortic reconstructive surgery. *British Journal of Anaesthesia* 1984; **56**: 465-71.
8. ALI HH, UTTING JE, GRAY TC. Stimulus frequency in the detection of neuromuscular block in humans. *British Journal of Anaesthesia* 1970; **42**: 967-78.
9. TASCH MD. Neuromuscular blocking agents in geriatric anesthesia. *International Anesthesiology Clinics* 1988; **26**: 152-5.
10. KENT AP, PARKER CJR, HUNTER JM. Pharmacokinetics of atracurium and laudanosine in the elderly. *British Journal of Anaesthesia* 1989; **63**: 661-6.
11. HARPER NJN, BRADSHAW EG, HEALEY TEJ. Evoked electromyographic and mechanical responses of the adductor pollicis compared during the onset of neuromuscular blockade by atracurium or alcuronium, and during antagonism by neostigmine. *British Journal of Anaesthesia* 1986; **58**: 1278-84.
12. DONATI F. Onset of action of relaxants. *Canadian Journal of Anaesthesia* 1988; **35**: S52-S58.
13. BELL PF, MIRAKHUR RK, CLARKE RSJ. Dose-response studies of atracurium, vecuronium and pancuronium in the elderly. *Anaesthesia* 1989; **44**: 925-7.
14. BLACKBURN CL, MORGAN M. Comparison of speed of onset of fazadinium, pancuronium, tubocurarine and suxamethonium. *British Journal of Anaesthesia* 1978; **50**: 361-4.
15. KEENS SJ, HUNTER JM, SNOWDON SL, UTTING JE. Potentiation of the neuromuscular blockade produced by alcuronium with halothane, enflurane and isoflurane. *British Journal of Anaesthesia* 1987; **59**: 1011-16.
16. DRIESSEN JJ, VREE TB, VAN EGMOND J, BOOIJ LDHJ, CRUL JF. Interaction of midazolam with two non-depolarizing neuromuscular blocking drugs in the rat *in vivo* sciatic nerve-tibialis anterior muscle preparation. *British Journal of Anaesthesia* 1985; **57**: 1089-94.

## Comparison of lumbar plexus block versus conventional opioid analgesia after total knee replacement

M. G. SERPELL, F. A. MILLAR AND M. F. THOMSON

### Summary

A randomised controlled study was undertaken to assess the analgesic efficacy of continuous lumbar plexus block for the first 48 hours after total knee replacement surgery. Boluses of 0.5% bupivacaine with adrenaline 1 in 200 000 (0.3 ml/kg) were administered through a cannula inserted into the neurovascular sheath of the femoral nerve. Thirteen patients who received this block required significantly less morphine than a control group of 16 patients. Pain scores were similar and there were no complications related to this technique.

### Key words

Anaesthetic techniques, regional; lumbar plexus block.

Anaesthetics, local; bupivacaine.

Analgesia, postoperative.

Postoperative pain is difficult to treat effectively, as suggested by the many techniques available,<sup>1</sup> but regional analgesia avoids many of the complications associated with conventional methods of treatment. Femoral nerve block provides good analgesia for open knee surgery,<sup>2</sup> and our practice is to repeat it as required for pain relief, but this relies on the availability of an experienced person to perform the block. The '3 in 1 block'<sup>3</sup> is a simple technique to block the femoral, lateral cutaneous nerve of thigh and obturator nerves, and the placement of an indwelling catheter allows continuous block of the lumbar plexus; this has been used successfully for pain relief after knee surgery.<sup>4,5</sup>

The purpose of this study was to evaluate the analgesic effect of continuous lumbar plexus block after total knee replacement.

### Methods

We studied patients of ASA grade 1 or 2, aged between 18 and 85 years, undergoing elective total knee replacement under spinal anaesthesia. Patients were not studied if they had a history of neurological disease, hypersensitivity to bupivacaine or were unsuitable for spinal anaesthesia. The study was approved by the hospital Ethics Committee. A full explanation of the On-Demand Analgesic Computer (ODAC, Janssen Scientific Instruments, Beerse, Belgium) was given and informed consent obtained.

The anaesthetic technique was standardised and comprised temazepam premedication 0.3 mg/kg to the nearest 10 mg orally 1 hour before surgery. All patients underwent spinal anaesthesia with 0.75% bupivacaine 2.75–3.25 ml. Peri-operative oxygen therapy was administered and sedation provided with either intravenous midazolam in 1–2-mg increments or a propofol infusion as required. No supplementary analgesia was administered.

Patients were allocated randomly into two groups. Group A had an 18-gauge cannula threaded over a 22-gauge metal spinal needle which was placed accurately into the femoral nerve sheath using a nerve stimulator. The minimum current required to elicit a contraction in the quadriceps muscle was used.

The block was performed immediately after surgery while the effects of the spinal were still present. The cannula was connected, after withdrawal of spinal needle, to a short extension tube and bacterial filter. The insertion site was then covered with a sterile occlusive dressing.

A dose of 0.3 ml/kg 0.5% bupivacaine with adrenaline 1:200 000 was administered in 5-ml increments following aspiration, while observing cardiovascular parameters to further exclude intravenous injection. The cannula was left *in situ* to enable top-ups of the same dose of bupivacaine at 6–8 hour intervals during the next 48 hours.

Group B did not receive this block nor did they have a placebo injection at the femoral nerve site. Both groups

M.G. Serpell, MB, ChB, FCAnaes, F.A. Millar, MB, ChB, Registrars, M.F. Thomson, MB, ChB, FFARCSI, Consultant, Department of Anaesthesia, Ninewells Hospital and Medical School, Dundee DD1 9SY

Accepted 2 July 1990.

were attached to the ODAC soon after returning to the ward and analgesic requirements were recorded by means of a printer. The ODAC was set to deliver morphine in 2-mg intravenous boluses to a maximum of 12 mg/hour with a lockout interval of 9 minutes. If pain relief was inadequate, intramuscular morphine 10 mg or paracetamol 1 g orally was administered.

The patient's assessment of postoperative pain was evaluated at 24 and 48 hours after surgery using a linear analogue on a scale from 0 to 100 (0, no pain, 100, worst pain imaginable). Any adverse effects were also noted at these times.

Results were analysed statistically by Student's *t*-test, Chi-squared test with Yates' correction or two-tailed Wilcoxon test as appropriate.

### Results

Thirty patients entered the trial, but one in Group A was withdrawn because of postoperative urinary retention which required bouginage under sedation and local anaesthetic to dilate a urethral stricture. Patient data, duration of surgery and peri-operative sedation requirements are summarised in Table 1. Two in each group received a propofol infusion for sedation at 2–3 mg/kg/hour.

Patients with the lumbar plexus block required a third less morphine than the control group in the 48 hour postoperative period ( $p < 0.05$ ). The ODAC system provided most of the analgesia because the doses of intramuscular morphine and oral paracetamol were low in both groups (Table 2).

Postoperative pain scores at 24 and 48 hours were similar, even though Group B had twice the number of refused ODAC requests compared to Group A. The incidence of nausea and vomiting were comparable, and although there was less antiemetic administered to the study group, this did not reach significance (Table 3).

### Discussion

We have demonstrated a significant reduction in postoperative analgesic requirements by using a continuous femoral nerve block ( $p < 0.05$ ). Use of the ODAC system provides a good indication of the magnitude of pain since the demand rate reflects a balance between attaining analgesia and avoiding side effects. The similar mean pain scores and incidence of nausea and vomiting in both groups supports this. We found, despite training the patients to use the ODAC as soon as pain was appreciated, that when the spinal analgesia wore off, it did so very rapidly and the ODAC could not deliver a large enough

**Table 2.** Postoperative analgesic requirements, mean (SEM). \* $p < 0.05$  (Wilcoxin two-tailed test).

	Group A ( <i>n</i> = 13)	Group B ( <i>n</i> = 16)
Total 48 hour consumption of morphine; mg	60 (8.7)*	91 (9.8)
Number of refused ODAC requests in 48 hours	6.2 (1.4)	12.9 (2.6)
Total number of intramuscular supplements; 10 mg	10	17
Range of frequency of administration of morphine supplements; mean	0–3 (0.8)	0–5 (1.1)
Total number of paracetamol supplements; 1 g	13	10
Range of frequency of administration of paracetamol supplements; mean	0–4 (1)	0–2 (0.6)

loading dose of morphine. Patients would often request an intramuscular supplement, and were then able to control pain by a maintenance demand rate.

The knee joint is innervated by components of the femoral, obturator and sciatic nerves. Only the first two are involved in the lumbar plexus block so complete analgesia is unlikely. Quadriceps muscle spasm aggravates pain in the knee joint (femoral nerve) and abolition of this muscle spasm by femoral nerve blockade may well be the main benefit of this analgesic technique. Other effective regional techniques used for postoperative analgesia include epidural opioids and (or) local anaesthetics. The complications of urinary retention, pruritus and respiratory depression from epidural opioids requires close observation of the patient for at least 24 hours. Epidural local anaesthetics provide the most effective analgesia but the bilateral motor blockade makes the patient more nurse dependent and the sympathetic blockade necessitates cardiovascular monitoring. Several studies have shown the benefits of the '3 in 1' block in patella, knee ligament surgery and arthroscopy,<sup>4–6</sup> but Dahl stated there was no advantage when used in knee arthroplasty.<sup>7</sup> There were only six patients in each of his groups and the block was performed after operation. There is evidence<sup>8</sup> that abolishing sensory input before the occurrence of painful stimuli can reduce postoperative analgesic requirements and we performed the block before spinal anaesthesia wore off.

Our study was not double-blind, because we thought it was unethical to submit a patient to a placebo which involved an invasive procedure. Secondly, any trial that

**Table 1.** Demographic data, mean (SEM). No significant differences ( $p < 0.05$ ) were found (Student's *t*-test).

	Group A ( <i>n</i> = 13)	Group B ( <i>n</i> = 16)
Males/females	2/11	4/12
Age; years	68 (2.7)	70 (2.5)
Height; cm	160 (1.5)	165 (3.0)
Weight; kg	70 (2.3)	72 (3.8)
Operation time; minutes	105 (5.1)	105 (4.8)
Midazolam; mg	4.0 (0.9)	2.9 (0.5)
(n = 11 and 14 respectively)		

**Table 3.** Pain scores (0 = no pain, 100 = worst pain imaginable), incidence of side effects and antiemetic administration, mean (SEM). Chi-squared and Wilcoxon test.

	Group A ( <i>n</i> = 13)	Group B ( <i>n</i> = 16)
Pain score; 24 hours	32 (8.8)	40 (5.5)
Pain score; 48 hours	34 (7.6)	37 (6.5)
Nausea; number of patients	9	12
Vomiting; number of patients	7	8
Total number of doses of prochlorperazine; 12.5 mg	11	24
Range of frequency of prochlorperazine administrations; mean	0–2 (0.8)	0–3 (1.5)



involved local anaesthetic drugs will produce paraesthesia and weakness which is obvious to both doctor and patient. It was difficult to assess for paraesthesia over the distribution of the affected nerves because of extensive dressings around the leg. Previous studies have demonstrated that there is usually evidence of femoral nerve anaesthesia but this is variable for lateral cutaneous nerve of thigh and rare for the obturator nerve.<sup>5,9</sup>

The 6–8 hour top-up regimen was devised to be as workable as possible so that the resident doctors would not be called to administer a dose during 'unsociable' hours (2400–0800). This problem could be solved by setting up an infusion or allowing suitably trained nurses to give the top-ups.

We did not measure plasma concentration of bupivacaine, but group A received an average of 5.8 top-ups (range 3–8) or 0.18 mg/kg/hour (SEM 0.01) over the 48-hour period. This is lower than the amount given in another study<sup>5</sup> which demonstrated safe subtoxic concentrations, so there is scope for giving more frequent doses if required. There were no complications of prolonged anaesthesia or infection at the cannula site, although damage to the femoral nerve has been reported in isolated cases.<sup>10,11</sup> We used a nerve stimulator for accurate placement of the cannula, and it is suggested that the avoidance of eliciting paraesthesia during nerve blocks reduces the incidence of damage.<sup>12</sup>

In conclusion, we have demonstrated that continuous lumbar plexus blockade is a safe and simple technique to perform and provides similar analgesia to parenteral morphine, but with significantly reduced opioid requirements.

## References

1. DODSON ME. A review of methods for relief of postoperative pain. *Annals of the Royal College of Surgeons of England* 1982; **64**: 324–7.
2. TIERNEY E, LEWIS G, HURTIG JB. Femoral nerve block with bupivacaine 0.25 per cent for postoperative analgesia after open knee surgery. *Canadian Journal of Anaesthesia* 1987; **34**: 455–8.
3. WINNIE AP, RAMAMURTHY S, DURANNI Z. The inguinal paravascular technic of lumbar plexus anesthesia: The '3-in-1' block'. *Anesthesia and Analgesia* 1973; **52**: 989–96.
4. ROSENBLATT RM. Continuous femoral anesthesia for lower extremity surgery. *Anesthesia and Analgesia* 1980; **59**: 631–2.
5. DAHL JB, CHRISTIANSEN CL, DAUGAARD JJ, SCHULTZ P, CARLSSON P. Continuous blockade of the lumbar plexus after knee surgery—postoperative analgesia and bupivacaine plasma concentrations. A controlled clinical trial. *Anaesthesia* 1988; **43**: 1015–8.
6. PATAL NJ, FLASHBURG MH, PASKIN S, GROSSMAN R. A regional anesthetic technique compared to general anesthesia for outpatient knee arthroscopy. *Anesthesia and Analgesia* 1986; **65**: 185–7.
7. DAHL JB, ANKER-MOLLER E, SPANSBERG N, SCHULTZ P, CHRISTENSEN EF. Continuous lumbar plexus block after arthroplasty. *Anaesthesia* 1988; **43**: 989.
8. RINGROSE NH, CROSS MJ. Femoral nerve block in knee joint surgery. *American Journal of Sports Medicine* 1984; **12**: 398–402.
9. MADEJ TH, ELLIS FR, HALSALL PJ. Evaluation of '3 in 1' lumbar plexus block in patients having muscle biopsy. *British Journal of Anaesthesia* 1989; **62**: 515–7.
10. JOHR M. Spaete komplikation der kontinuierlichen blockade des n. femoralis. *Regional Anaesthesia* 1987; **10**: 37–8.
11. NIELSEN BF. Iatrogenic femoral nerve neuropathy after lumbar plexus block. *Ugeskrift for Laeger* 1988; **150**: 748–9.
12. SELANDER D, EDSHAGE S, WOLFF T. Paresthesiae or no paresthesiae. Nerve lesions after axillary blocks. *Acta Anaesthesiologica Scandinavica* 1979; **23**: 27–33.

## Addition of fentanyl to prilocaine for intravenous regional anaesthesia

P. ARMSTRONG, I. POWER AND J. A. W. WILDSMITH

### Summary

*Fifteen volunteers underwent intravenous regional anaesthesia on two occasions using 40 ml 0.5% prilocaine, to which had been added either 2 ml 0.9% saline or 0.1 mg fentanyl (resultant concentration 2.5 µg/ml). There was no difference in the rate of onset of blockade of cold sensation from an ethyl chloride spray, or to sharp and touch pinprick sensation in either group. There was an increase in the incidence of nausea after tourniquet release in the fentanyl group. It is concluded that the addition of fentanyl 2.5 µg/ml to prilocaine 0.5% confers no benefit in intravenous regional anaesthesia.*

### Key words

Anaesthetics, local; prilocaine.

Analgesics; fentanyl.

Anaesthetic techniques, regional; intravenous.

Local anaesthetics act by blocking conduction in peripheral nerve axons. In contrast, the antinociceptive action of opioids is believed to be because of interaction with receptors in the central nervous system. Fentanyl, a synthetic opioid of the phenylpiperidine series is used widely peri-operatively as a parenteral analgesic. In addition, it has also been used epidurally for both postoperative pain relief<sup>1</sup> and in obstetrics.<sup>2,3</sup> Its analgesic action is believed to follow diffusion of the drug through the dura mater and CSF and thence interaction with the opioid receptors in the substantia gelatinosa.

However, *in vitro* experiments on isolated desheathed rabbit vagal nerve preparations have shown that fentanyl has a local anaesthetic action<sup>4</sup> and that it augments the effects of bupivacaine.<sup>5</sup> The threshold concentration for this *in vitro* effect appears to be about 50 µg/ml, but the addition of 0.1 mg fentanyl to 30 ml 1.5% lignocaine with adrenaline 1:200 000 (fentanyl 3.3 µg/ml) produced a significantly more intense sensory and motor block after 5 minutes in axillary plexus blockade in man. The block also had a significantly longer duration of action,<sup>6</sup> but it is possible that both effects were because of the central analgesic action of fentanyl rather than any local effect.

Intravenous regional anaesthesia (IVRA) isolates the arm from the rest of the circulation and is therefore a useful model for studying the peripheral actions of a drug in the absence of central effects. This study was designed to investigate the effects of the addition of fentanyl (2.5 µg/ml) to 0.5% prilocaine during IVRA in volunteers.

### Subjects and methods

Fifteen healthy, fasted, male volunteers aged 27–35 years acted as subjects in this study, which received approval from the local hospital ethics committee. Each subject underwent IVRA on their nondominant upper limb on two occasions, separated by at least 4 days.

On both occasions, a standard technique was used. Two 22-G cannulae were inserted, one into a prominent vein on the ulnar side of the dorsum of the hand to be injected (the same site being used on both occasions) and the other in the contralateral hand. A padded pneumatic tourniquet was placed around the upper arm at its thickest point and the arm exsanguinated with an Esmarch bandage. The same tourniquet site was used on both occasions. The tourniquet was inflated to 250 mmHg (at least 100 mmHg about the systolic blood pressure for all subjects) and the bandage removed.

Forty-two ml of solution was then injected over 30 seconds. The solution consisted of either 40 ml 0.5% prilocaine hydrochloride to which was added 2 ml 0.9% saline (saline group) or 0.1 mg fentanyl so that the resultant concentration was approximately 2.5 µg/ml (fentanyl group). The solutions were administered in a double-blinded, randomised (from a table of random numbers) fashion. The solutions were prepared by a second anaesthetist so that the nature of the additive was unknown to either the subject or investigator. Time zero was the end of injection. Thereafter sensation was tested at 1-minute inter-

P. Armstrong, MB, ChB, FCAnaes, Research Fellow, I. Power, MB, ChB, FCAnaes, Lecturer, J.A.W. Wildsmith, MD, FCAnaes, Consultant, Department of Anaesthesia, University of Edinburgh, Royal Infirmary Edinburgh, Lauriston Place, Edinburgh EH3 9YW.

Accepted 19 June 1990.

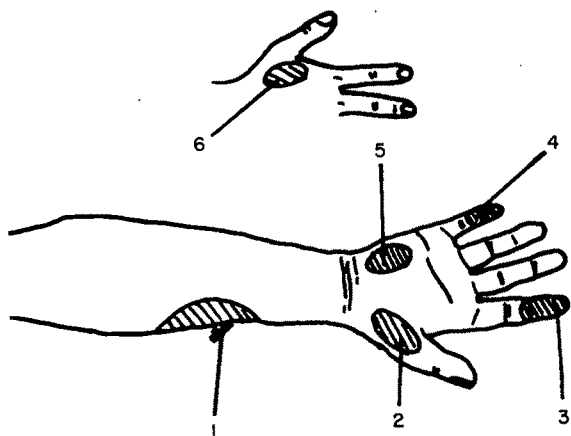


Fig. 1. Sites chosen for sensory testing. 1. forearm (radial nerve); 2. thenar eminence (median nerve); 3. index finger (median nerve); 4. little finger (ulnar nerve); 5. hypothenar eminence (ulnar nerve); 6. first webspace (radial nerve).

vals. A short-bevelled needle was used; the subject reported the sensation as sharp, touch or absent. Temperature sensation was tested using a small amount of ethyl chloride spray; the sensation was reported as cold or absent. Six sites were assessed (Fig. 1) chosen for their representation of small peripheral nerve branches.<sup>7</sup> The subject was unable to see the arm being tested.

Twenty minutes after time zero, the tourniquet was released and sensation testing continued until full recovery had occurred. The subjects reported any systemic sensation experienced after cuff release. Naloxone 0.4 mg was injected if the subject felt dizzy. The respiratory rate was noted 5 minutes before and after cuff release.

The results were analysed for each of the sensation modalities tested (pinprick, touch and cold) using the Wilcoxon signed rank test. The frequency of systemic sequelae experienced were analysed using a Chi-squared table. A *p* value of less than 0.05 was accepted as significant.

### Results

All subjects lost both cold and pinprick sensation at all six sites within the 20-minute time that the tourniquet was inflated during both blocks. In contrast, two individuals in the fentanyl group and three in the saline group still had some touch pinprick sensation remaining at 20 minutes. There was no difference between the groups in the speed of

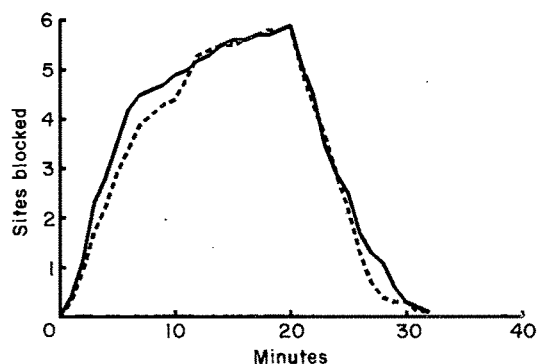


Fig. 2. Time course to onset and recovery of blockade to touch; — 100 µg fentanyl; - - - saline.

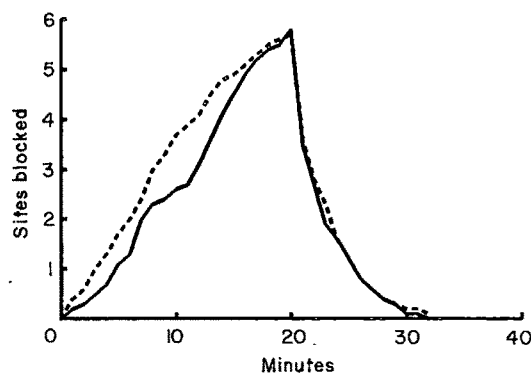


Fig. 3. Time course to onset and recovery of blockade to sharp pinprick; — 100 µg fentanyl; - - - saline.

onset of blockade of any of the sensory modalities (Figs 2-4).

Cold sensation was lost more quickly than sharp pinprick. The loss was statistically faster for the first 15 minutes and thereafter the loss was similar. Touch sensation was less affected at all time intervals. After cuff release, sensation rapidly returned at all six sites with virtually total recovery after 10 minutes. There was no difference between the two groups in the speed of recovery.

Both nausea and dizziness were experienced soon after cuff release. Three different subjects in both groups were dizzy, seven were nauseated in the fentanyl group and one in the saline group ( $p < 0.02$ , Chi-squared test). All recovered fully from the nausea on injection of naloxone, although this had no effect on the dizziness. There was no change in the respiratory rate in either group following tourniquet release.

### Discussion

*In vitro* studies on the isolated rabbit vagus nerve have shown that fentanyl will reversibly inhibit nerve impulse transmission in a concentration dependent manner in unsheathed, but not sheathed nerves.<sup>4</sup> This implies that fentanyl is not able to penetrate intact nerves, but that the nerve endings at which IVRA is believed to have its initial site of action<sup>7,8</sup> should be accessible.

Given its *in vitro* potency, it was thought unlikely that the addition of 2.5 µg/ml fentanyl would have a significant effect, but the same amount (0.1 mg) of fentanyl had an effect in axillary nerve blockade<sup>6</sup> and not a dissimilar dose

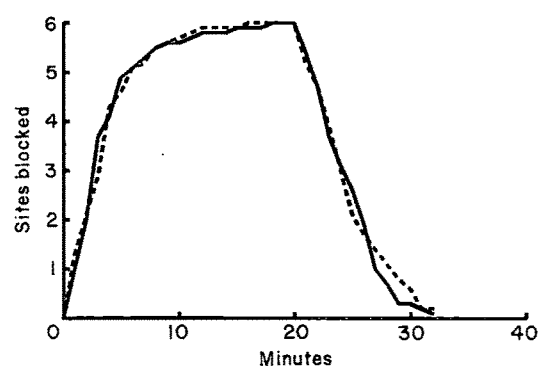


Fig. 4. Time course to onset and recovery of blockade to cold; — 100 µg fentanyl; - - - saline.

(25 µg in 4 ml water) was found to provide good analgesia after unilateral lumbrosacral plexus injection.<sup>9</sup>

The results show that this concentration of fentanyl is without significant local action in man. Use of the isolated forearm technique should prevent fentanyl from reaching the central nervous system and therefore prevent receptor stimulation. Some solution may leak past the cuff,<sup>10</sup> although the amount in this trial should have been small because the injection was both slow and distal and the arm exsanguinated.<sup>11</sup> In addition, no subject felt nauseated during the time the tourniquet was inflated. The use of a higher concentration might have an obvious local action, but would be associated with much greater central effects because the total dose injected would be very high. Even with a total dose of 100 µg the incidence of systemic effects was significant and thus could not be increased.

It is therefore likely that addition of fentanyl to local anaesthetics *in vivo* has no direct effect upon nerve conduction, but probably has its analgesic effects via interaction with central opiate receptors. This might also explain the effects seen after lumbrosacral injection,<sup>9</sup> but an additional factor in that situation was the hypotonicity of the solution used. This might have its own nonspecific effects on nerve conduction.

It is interesting that temperature sensation was lost faster than pinprick sensation in both groups whilst touch sensation was most resistant to blockade. Touch sensation is transmitted by Aβ fibres, which being more myelinated may be less sensitive to block than either temperature or pain (Aδ and C).<sup>12</sup> This finding is contrary to a similar study<sup>13</sup> and to other work showing that temperature and pain sensations were blocked at a similar rate.<sup>14</sup>

In conclusion, fentanyl 2.5 µg/ml has no potentiating effect on the onset or duration of nerve blockade in IVRA, but it did cause an increase in nausea. Potentiation of blockade produced by the addition of fentanyl to local anaesthetics *in vivo* therefore appears to be a central effect. If such an effect is required during peripheral nerve block techniques, it is suggested that it is more appropriate to inject the drug systemically and titrate its effect.

### Acknowledgments

Dr P. Armstrong is the research fellow of the Association of Anaesthetists of Great Britain and Ireland.

### References

1. LOMESSEY A, MAGNIN C, VIALE JP, MOTIN J, COHEN R. Clinical advantages of fentanyl given epidurally for post-operative analgesia. *Anesthesiology* 1984; **61**: 466–9.
2. VELLA LM, WILLATS DG, KNOTT C, LINTIN DJ, JUSTINS DM, REYNOLDS F. Epidural fentanyl in labour. *Anaesthesia* 1985; **40**: 741–7.
3. NAULTY JS, DATTA S, OSTHEIMER GW, JOHNSON MD, BURGER GA. Epidural fentanyl for postcesarean delivery pain management. *Anesthesiology* 1985; **63**: 694–8.
4. GISSEN AJ, GUGINO LD, DATTA S, MILLER J, COVINO BG. Effects of fentanyl and sufentanil on peripheral mammalian nerves. *Anesthesia and Analgesia* 1987; **66**: 1272–1276.
5. POWER I, BROWN DT, WILDSMITH JAW. Effect of fentanyl on nerve conduction. *British Journal of Anaesthesia* 1988; **61**: 507P–508P.
6. GOBEAUX D, LANDAIS A, BEXON G, CAZABAN J, LEVRON JC. Adjonction de fentanyl à la lidocaine adrénalinée pour le blocage du plexus brachial. *Cahiers d'Anesthesiologie* 1987; **35**: 195–9.
7. URBAN BJ, MCKAIN CW. Onset and progression of intravenous regional anesthesia with dilute lidocaine. *Anesthesia and Analgesia* 1982; **61**: 834–8.
8. RAJ PP, GARCIA CE, BURLESON JW, JENKINS MT. The site of action of intravenous anesthesia. *Anesthesia and Analgesia*... 1972; **51**: 776–786.
9. SMITH B, PINNOCK C, FISCHER B, TROTTER T, SCOTT P. Unilateral analgesia following injection of fentanyl into the lumbosacral plexus. *Lancet* 1987; **1**: 1497–8.
10. ROSENBERG PH, KALSO EA, TUOMINEN MK, LINDÉN HB. Acute bupivacaine toxicity as a result of venous leakage under the tourniquet cuff during a Bier block. *Anesthesiology* 1983; **58**: 95–8.
11. FINEGAN BA, BUKHT MD. Venous pressures in the isolated upper limb during saline injection. *Canadian Anaesthetists' Society Journal* 1983; **31**: 364–67.
12. WILDSMITH JAW. Peripheral nerve and local anaesthetic drugs. *British Journal of Anaesthesia* 1986; **58**: 692–700.
13. ARMSTRONG P, BROCKWAY M, WILDSMITH JAW. Alkalinisation of prilocaine for intravenous regional anaesthesia. *Anaesthesia* 1990; **45**: 11–13.
14. STRICHARTZ GR, ed. *Handbook of experimental pharmacology, Local anesthetics* Vol. 68 Heidelberg: Springer-Verlag, 1986.

## Vasovagal asystole during spinal anaesthesia

I. McCONACHIE

### Summary

*A patient in whom vasovagal asystole was induced by the performance of a spinal anaesthetic in the upright position is described. This illustrates the importance of routine monitoring of the electrocardiograph during regional anaesthesia. The risk of vasovagal syncope may be increased by the use of the sitting position and by the omission of anxiolytic premedication.*

### Key words

*Anaesthetic techniques, regional; spinal.  
Complications; asystole.*

### Case history

A 34-year-old man presented for internal fixation of his left ankle following a fracture. He was physically fit and history and examination were unremarkable. His ankle had been manipulated under general anaesthesia 2 days previously but the fracture alignment was unsatisfactory. The patient complained of nausea after his first anaesthetic, so a spinal anaesthetic was suggested as an alternative for the second procedure. There was a full discussion of risks and side effects. No premedication was given at the patient's request.

The arterial blood pressure was measured on arrival in the anaesthetic room (systolic 130 mmHg) and electrocardiograph (ECG) leads attached for continuous monitoring. After subcutaneous infiltration of 1% lignocaine, a 16-G cannula was inserted into a vein on the left forearm and 500 ml of a crystalloid solution was infused rapidly.

The patient was positioned on the trolley sitting upright with his head bent over his knees. Spinal anaesthesia was performed using an aseptic technique. Two ml 1% lignocaine were infiltrated subcutaneously over the L<sub>2-3</sub> interspace. A 25-G spinal needle was inserted through the skin and spinal ligaments at that level until clear cerebrospinal fluid (CSF) was obtained. Two and a half ml of 0.5% bupivacaine in dextrose was injected into the subarachnoid space. As the injection was being completed, the ECG trace was seen to be flat. The ECG electrodes were still attached to the patient but his eyes were closed, his breathing was stertorous and no peripheral pulse could be palpated. The needle was removed from the patient's back immediately, he was laid supine and oxygen was administered.

Spontaneous return of electrical activity occurred without any further therapeutic manoeuvres, the patient awoke and rapidly became alert. Arterial pressure was normal and remained so. A further infusion of crystalloid was given until a satisfactory level of spinal analgesia was achieved. The patient was now well, so the decision was made to proceed. The operation was uneventful and there were no postoperative sequelae attributable to the incident.

### Discussion

Vasovagal collapse may be due to vagal slowing of the heart with or without loss of peripheral vascular tone.<sup>1</sup> Fear, pain and an upright posture may all contribute.<sup>1</sup> Although the patient seemed calm, the sequence of events strongly suggests that the cause of asystole was a vasovagal episode rather than the spinal anaesthetic itself.<sup>2</sup> This is supported by the fact that when the patient was placed supine and given oxygen, normal cardiac rhythm returned and he rapidly recovered consciousness.

This case provides further evidence to support the routine use of continuous ECG monitoring in the anaesthetic room. This applies equally to patients receiving general and regional anaesthesia, although it has been suggested that frequently such monitoring does not occur.<sup>3,4</sup> In the absence of the initial warning from the ECG trace, this patient's loss of consciousness would not have been immediately apparent. His position, slumped over his knees, would not have allowed him to fall over in any direction, especially as he was being supported by the

I. McConachie, MB, ChB, FFARCS, Lecturer in Anaesthesia, University of Manchester, Hope Hospital, Salford M6 8HD.  
Accepted 4 August 1990.



anaesthetic nurse. If this position had been sustained during the period of asystole full recovery may have been less certain. This case illustrates a potential hazard of inducing spinal anaesthesia in the upright position. The cardiovascular effects of vasovagal bradycardia or asystole are accentuated in the upright position due to a reduction in venous return.<sup>1,5</sup> Vasovagal syncope has been described during spinal anaesthesia in the sitting position<sup>6</sup> although in these cases the ECG was not monitored, so asystole was not confirmed. In this patient the omission of an anxiolytic drug may have contributed, even though he appeared calm. Anxiety, even when denied, has been shown to be a major factor in vasovagal syncope during spinal anaesthesia.<sup>7</sup> It seems that even today, patients sometimes may literally be scared to death.<sup>8</sup>

#### References

1. GILLIAT RW. Syncope. In: WEATHERALL DJ, LEDINGHAM JGG, WARRELL DA, eds. *Oxford textbook of medicine* 2nd edn. Oxford: Oxford University Press 21.51–21.53.
2. CAPLAN RA, WARD RJ, POSNER K, CHENEY FW. Unexpected cardiac arrest during spinal anesthesia: a closed claims analysis of predisposing factors. *Anesthesiology* 1988; **68**: 5–11.
3. MATHIAS JA, LUNN JN. Minimal monitoring and vigilance. *Anaesthesia* 1987; **42**: 683–4.
4. LONG TMW. Asystole on induction: do we monitor it? *Anaesthesia* 1989; **44**: 354–5.
5. GLICK G, YU PN. Hemodynamic changes during spontaneous vasovagal reactions. *American Journal of Medicine* 1963; **34**: 42–51.
6. MORGAN C. Collapse during spinal anaesthesia? *Anaesthesia and Intensive Care* 1978; **6**: 169.
7. TOLKSDORF W, MERKEL G, REHDER H, REY ER, BERLIN J. Psychologische aspekte der spinalanaesthesie. *Anesthesist* 1984; **33**: 307–10. (Title in English: Psychological aspects of spinal anaesthesia. Vasovagal syncope and anxiolytic medication).
8. SCHRAEDER PL, PONTZER R, ENGEL TR. A case of being scared to death. *Archives of Internal Medicine* 1983; **143**: 1793–4.

CASE REPORT

## An unusual cause of obstructive emphysema

R. J. BUIST

### Summary

*A 6-month-old child presented with a history, physical signs and radiographic findings suggestive of the presence of a foreign body in the tracheobronchial tree. However, further investigation revealed extrinsic compression of the left main bronchus by a grossly enlarged left atrium. Treatment of the patient's heart failure resulted in resolution of the signs. Anomalous origin of the left coronary artery was found to be the cause.*

### Key words

*Complications; obstructive emphysema.*

Tracheobronchial foreign body aspiration is an important cause of morbidity in children and has a significant mortality.<sup>1</sup> Delayed diagnosis is an important factor in determining outcome.<sup>2,3</sup> Although only 4% of endobronchial foreign bodies are radiopaque, obstructive emphysema can be demonstrated in up to 60% of patients.<sup>4</sup> However, as this case illustrates, there are less common and more sinister causes of this radiological finding.

### Case history

A 6-month-old 7.6-kg male infant was admitted to the referring district general hospital with a 3-week history of cough, coryza and increasing dyspnoea. One week before admission he had had an episode of choking. The general practitioner had diagnosed a chest infection and prescribed amoxycillin. The child had no significant past medical history. On admission to the referring hospital he was peripherally cyanosed, with a respiratory rate of 50–60 breaths/minute, decreased air entry on the left side of his chest and had a 3 cm hepatomegaly. He was treated with 80% oxygen in a headbox. Arterial blood gas analysis showed a pH of 7.37,  $P_{aCO_2}$  3.7 kPa,  $P_{aO_2}$  24.9 kPa, bicarbonate 16 mmol/litre and base excess –7.1 mmol/litre. Other investigations revealed a haemoglobin of 9.3 g/dlitre, total white cell count  $8.5 \times 10^6$ /litre, urea 7.3 mmol/litre and creatinine 65 mmol/litre. A chest radiograph (Fig. 1) showed tracheal deviation to the right with hyperinflation of the left lung field and cardiomegaly.

He was transferred to Great Ormond Street with a differential diagnosis of inhaled foreign body, congenital lobar emphysema, extrinsic compression of the left main bronchus or a primary congenital cardiac abnormality. On arrival he was pyrexial (38.5°C), but was cold peripherally with a tachycardia of 180 beats/minute and an arterial blood pressure of 90/60 mmHg. He showed signs of respiratory distress, with a tachypnoea of 50 breaths/minute and marked subcostal recession with reduced air entry on the left side of the chest. Capillary blood gas analysis revealed a pH of 7.18,  $P_{aCO_2}$  7.9 kPa, bicarbonate 22.5 mmol/litre and base excess –7 mmol/litre. He was referred to the cardiothoracic surgeons for consideration for urgent bronchoscopy. In view of his cardiomegaly echocardiography was performed. This revealed a huge left atrium, a poorly contractile left ventricle and normal coronary arteries. Treatment with frusemide, dopamine, digoxin, sodium nitroprusside, erythromycin and gentamicin was commenced and 100 ml of human albumin solution was given. Oxygen 60% was administered via a headbox.

Despite treatment the patient deteriorated with increasing respiratory rate, subcostal recession, gasping respiration and poor peripheral perfusion. His trachea was intubated and his lungs gently ventilated. Arterial blood gas analysis immediately after intubation ( $F_{iO_2}$  1.0, on manual ventilation with a T-piece) showed a pH of 7.21,  $P_{aCO_2}$  7.4 kPa,  $P_{aO_2}$  16.2 kPa, bicarbonate 19.9 mmol/litre and base excess –6.4 mmol/litre. Following intubation and ventilation there was marked improvement both in the

R.J. Buist,\* BA, MBBS, FFARCS, Senior Registrar, Department of Anaesthesia, The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH.

\*Present appointment: Senior Registrar in Anaesthesia St Thomas' Hospital, Lambeth Palace Road, London SE1 7EH.  
Accepted 1 August 1990.

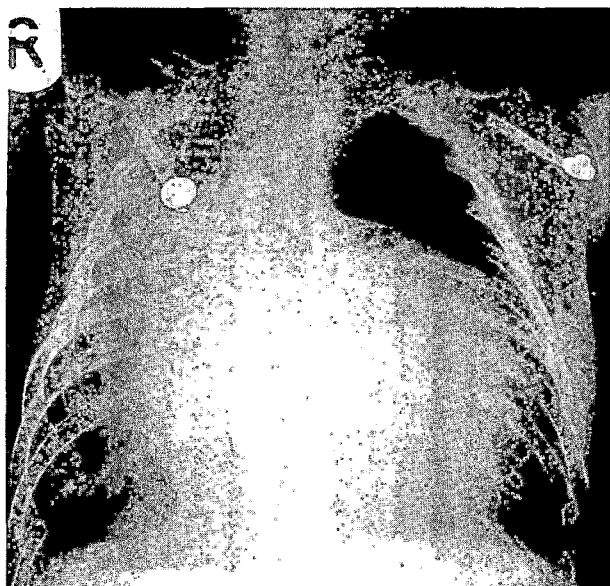


Fig. 1. Chest radiograph before tracheal intubation showing cardiac enlargement, tracheal deviation to the right and hyperlucency of the left lung.

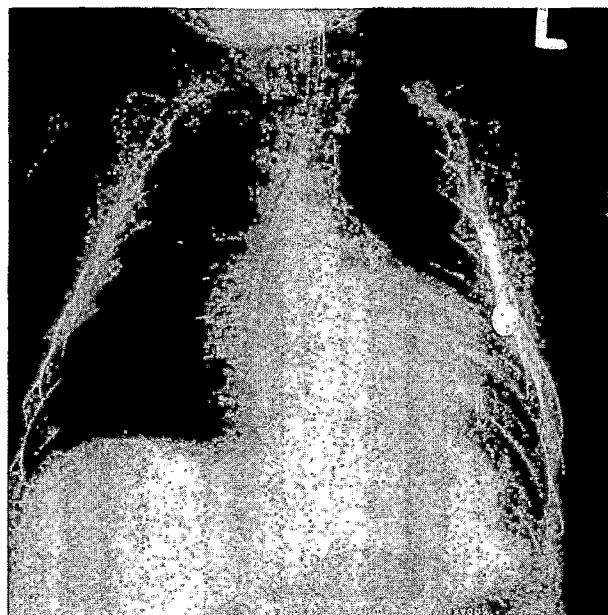


Fig. 2. Chest radiograph following tracheal intubation and treatment of cardiac failure.

clinical condition of the patient and in arterial blood gases (IPPV, pressures 30/4 cmH<sub>2</sub>O,  $F_{iO_2}$  0.4; pH 7.58,  $P_{aCO_2}$  3.9 kPa,  $P_{aO_2}$  19.5 kPa and base excess 7.6 mmol/litre).

Tracheal intubation and artificial ventilation were maintained for 7 days in total. Radiographically (Fig. 2) the cardiomegaly and emphysema decreased but repeat echocardiography showed a poorly contractile left ventricle (left ventricular end-diastolic diameter 29 mm, left ventricular end-systolic diameter 24 mm) despite vigorous treatment with frusemide, captopril, spironolactone and digoxin. Cardiac catheterisation was carried out in view of this persistent poor left ventricular function. This showed a large right coronary artery and an anomalous left coronary artery arising from the pulmonary artery. One month after admission re-implantation of the left coronary artery onto the aorta was carried out on cardiopulmonary bypass.

### Discussion

Obstructive emphysema in childhood may be due to intra-bronchial or extrabronchial obstruction.<sup>5</sup> The differential diagnosis includes congenital lobar emphysema (although 95% of these present before 6 months of age), bronchogenic cyst, external bronchial compression (from an abnormal blood vessel or lymph node), intrabronchial or mediastinal tumours and intrabronchial tuberculosis.<sup>5</sup> Intrinsic obstruction of the bronchus may result from bronchomalacia, bronchostenosis, redundant mucosal fold or alveolar fibrosis.<sup>6</sup> Nevertheless, the most common cause (between the ages of 6 months and 4 years) is aspiration of a foreign body into the tracheobronchial tree and the associated inflammatory reaction.<sup>5</sup>

There were several features of this case which were atypical for a diagnosis of foreign body aspiration. In one published series 85% of patients with foreign body aspi-

ration were less than 3 years old and only one patient was aged 6 months.<sup>4</sup> There was no clear history of foreign body aspiration in this present patient. The chest radiograph showed clear evidence of cardiac enlargement which is not usually a feature of foreign body aspiration. Finally the patient's clinical condition and radiographic evidence of obstructive emphysema improved with artificial ventilation and treatment of cardiac failure.

However, as in this patient massive enlargement of the left atrium from a number of congenital cardiac lesions can cause external compression of the left main bronchus<sup>7</sup> and a check-valve type of obstruction.<sup>8</sup> Enlargement of the left atrium in this case was the result of severe cardiac failure due to anomalous origin of the left coronary artery from the pulmonary artery.

### References

1. LLOYD-THOMAS AR, BUSH GH. All that wheezes is not asthma. *Anaesthesia* 1986; **41**: 181-5.
2. PYMAN C. Inhaled foreign bodies in childhood: a review of 230 cases. *Medical Journal of Australia* 1971; **1**: 62-8.
3. ESLAMADO RM, RICHARDSON MA. Laryngotracheal foreign bodies in children. A comparison with bronchial foreign bodies. *American Journal of Diseases of Children* 1987; **141**: 259-62.
4. BLAZER S, NAVEH Y, FRIEDMAN A. Foreign body in the airway: A review of 200 cases. *American Journal of Diseases of Children* 1980; **134**: 68-71.
5. STERN RC. The respiratory system. In: BEHRMAN RE, VAUGHAN VC, NELSON W. eds. *Nelson textbook of pediatrics*, 12th edn. Philadelphia: W.B. Saunders, 1983: 1038-40.
6. LEAPE LL, HOLDER TM. Pediatric surgery. In: SABISTON DC, ed. *Davis-Christopher textbook of surgery*. Philadelphia: W.B. Saunders, 1981: 1358-9.
7. ARCINIEGAS E. Coronary artery anomalies. In: ARCINIEGAS E, ed. *Pediatric cardiac surgery*. Chicago: Yearbook Medical Publishers 1985: 389-402.
8. CHATTERJI S, CHATTERJI P. The management of foreign bodies in air passages. *Anaesthesia* 1972; **27**: 390-5.

CASE REPORT

## Milrinone, a bridge to heart transplantation

D. M. WATSON, K. M. SHERRY AND G. A. WESTON

### Summary

*We describe the use of a milrinone infusion as a bridge to heart transplantation in the treatment of severe congestive cardiac failure secondary to viral cardiomyopathy. The clinical improvement that occurred when milrinone was commenced made it possible to wean the patient from conventional cardiovascular support with an intra-aortic balloon pump and dobutamine. There was an improvement in organ perfusion.*

### Key words

Pharmacology; milrinone.

Surgery; heart transplantation.

This case report describes the use of the inodilator milrinone in a patient with end-stage congestive cardiac failure due to viral cardiomyopathy.

### Case history

A 52-year-old male with a 7-year history of viral cardiomyopathy was admitted to hospital with increasing dyspnoea. Drug therapy was with amiloride 10 mg twice daily, frusemide 250 mg once daily and captopril 50 mg three times per day. Diltiazem (60 mg three times a day) and warfarin were added to this regimen. His condition deteriorated and 5 days after admission inotropic support was started using dobutamine 7.5 µg/kg/minute and dopamine 2.5 µg/kg/minute. His condition continued to deteriorate and he was transferred to this centre for urgent heart transplantation.

On arrival the patient was in severe congestive cardiac failure, with a pulse rate of 96 beats/minute, arterial blood pressure 90/70 mmHg and a jugular venous pulse of 6 cmH<sub>2</sub>O. He was tachypnoeic and had widespread bilateral inspiratory crepitations. Arterial blood gases (*F*<sub>IO<sub>2</sub></sub> 0.5) revealed pH 7.53, *P*<sub>aO<sub>2</sub></sub> 10.89 kPa, *P*<sub>aCO<sub>2</sub></sub> 2.48 kPa and *S*<sub>aO<sub>2</sub></sub> 97%.

An isosorbide infusion was commenced at 3 mg/hour and supplemental doses of frusemide were given. This resulted in an initial clinical improvement and his urine output increased to 100–150 ml/hour. After 48 hours he again became increasingly breathless and unresponsive to

diuretics. The dose of dobutamine was increased to 15 µg/kg/minute and metolazone was added to the diuretic therapy. There was some clinical improvement in his condition with an increase in urinary output. However, 48 hours later he again deteriorated and was transferred to the cardiac intensive care unit. An intra-aortic balloon pump with maximum augmentation was added to his therapy. Following this his arterial blood pressures were: augmented 110 mmHg, systolic 105 mmHg, mean 90 mmHg and diastolic 65 mmHg. Arterial blood gas analysis (*F*<sub>IO<sub>2</sub></sub> 0.5) with spontaneous ventilation were pH 7.47, *P*<sub>aO<sub>2</sub></sub> 10.11 kPa, *P*<sub>aCO<sub>2</sub></sub> 3.95 kPa and *S*<sub>aO<sub>2</sub></sub> 96%. There was an initial improvement in urine output.

Twenty-four hours later he became increasingly confused and drowsy and developed a Cheyne Stokes pattern of respiration and a tachycardia of 133 beats/minute. His arterial pressures decreased to an augmented pressure of 93 mmHg and systolic pressure of 85 mmHg. The arterial blood gases with an *F*<sub>IO<sub>2</sub></sub> of 1.0 were pH 7.5, *P*<sub>aO<sub>2</sub></sub> 8.64 kPa, *P*<sub>aCO<sub>2</sub></sub> 4.0 kPa and *S*<sub>aO<sub>2</sub></sub> 90%.

A decision was made to commence inodilator therapy with milrinone. A loading dose of 50 µg/kg was given intravenously over a 10-minute period, followed by an infusion at the rate of 0.5 µg/kg/minute. There was no decrease in the augmented arterial pressure or the mean perfusion pressure. His mental state improved over the next 12 hours and he became alert and cooperative. His pulse rate decreased to 125 beats/minute and his arterial pressures improved to augmented 110 mmHg, systolic

D.M. Watson, FFARCSI, Senior Registrar, K.M. Sherry, FFARCS, Consultant, G.A. Weston, FFARCS, Consultant, Northern General Hospital, Herries Road, Sheffield S5 7AV.

Accepted 18 July 1990.

Table 1. Biochemistry before heart transplantation.

	26 February	12 March	15 March	17 March	19 March	26 March	28 March	2 April
Urea; mmol/litre	8.3	11	14.5	10.9	6.7	24.7	22.4	24.4
Creatinine; mmol/litre	136	137	153	122	103	141	165	136
Bilirubin; $\mu$ mol/litre	40	38	48	40	32	36	29	28
AST; IU/litre	118	124	130	129	112	70	72	65
ALT; IU/litre	230	247	251	257	165	122	112	108

\* Milrinone commenced 16 March.

105 mmHg, mean 90 mmHg and diastolic 70 mmHg. There was an increase in the urinary output in response to supplements of frusemide. Forty-eight hours later arterial blood gases ( $F_{IO_2}$  0.4) were pH 7.46,  $P_{aO_2}$  10.6 kPa,  $P_{aCO_2}$  4.3 kPa and  $S_{aO_2}$  92% and the patient was once again starting to take oral nutrition.

The patient was maintaining a systolic arterial pressure of 100 mmHg after 5 days, a urine output of over 100 ml/hour and a daily negative fluid balance of 1.5–2 litres. Biochemical analysis showed an improvement in renal and hepatic function (Table 1). The intra-aortic balloon pump was removed and the patient maintained a systolic blood pressure of 90–95 mmHg with a satisfactory urine output. He was transferred back to the ward at this time.

The dobutamine infusion was stopped 72 hours later and therapy was continued with milrinone 0.5  $\mu$ g/kg/minute, dopamine 2.5  $\mu$ g/kg/minute and isosorbide 3 mg/hour. Biochemically the patient's renal function gradually deteriorated in the period before transplantation although at this stage fluid was restricted and diuretic therapy was continued. The bilirubin, aspartate amino transferase (AST) and alanine aminotransferase (ALT) all continued to decrease indicating a steady improvement in hepatic function. The patient underwent heart transplant surgery 17 days after the milrinone was started. His postoperative recovery was satisfactory and he was discharged from hospital 28 days later.

### Discussion

Milrinone is a new phosphodiesterase III inhibitor with both inotropic and vasodilating properties; there are both oral and intravenous preparations. Intravenous milrinone increases myocardial contractility<sup>1</sup> in patients with severe congestive cardiac failure without increasing regional myocardial oxygen consumption.<sup>2</sup> The haemodynamic effects of milrinone are an increase in cardiac index with decreases in systemic and pulmonary vascular resistance and right atrial, pulmonary artery and pulmonary capillary wedge pressures. Heart rate is unchanged or increased and mean arterial pressure is unchanged or reduced.<sup>3</sup>

In this patient with longstanding congestive cardiac failure milrinone was added to the established  $\beta$  agonist and intra-aortic balloon pump therapy. We saw a clinical improvement in his condition and he had biochemical evidence of improved renal and hepatic function.

The haemodynamic effects of milrinone do not depend on catecholamine receptor stimulation but on inhibition of phosphodiesterase activity leading to increased cyclic AMP levels.<sup>4</sup> In addition animal studies suggest that milrinone increases calcium entry into cells and at high concentrations inhibits calcium uptake into the sarcolemma

membranes.<sup>5</sup> This increases the cytoplasmic calcium available for binding to actin and myosin filaments during myocardial contraction.

Patients with longstanding cardiac failure and those receiving long-term catecholamine therapy have reduced numbers of receptors and so have a reduced response to  $\beta$  agonist drugs.<sup>6</sup> The use of a phosphodiesterase inhibitor in these patients is an alternative to increase myocardial contractility. The clinical haemodynamic response to milrinone in this patient was impressive and sustained such that both  $\beta$  agonists and intra-aortic balloon pump could be withdrawn.

A single oral dose of milrinone increased renal blood flow in a study in patients with congestive cardiac failure,<sup>7</sup> although a longer study showed no change in renal blood flow, renal vascular resistance or glomerular filtration rate after one month of treatment.<sup>8</sup> Milrinone has no effect on hepatic blood flow.<sup>9</sup> There was an initial improvement in the biochemical indices of renal function in this patient, but a subsequent increase in creatinine and urea. His liver function biochemistry steadily improved. We believed that the initial improvement in renal biochemistry was due to his haemodynamic improvement and the later deterioration the result of aggressive diuretic therapy. This impression was substantiated by the sustained improvement in liver function tests which more closely followed his haemodynamic improvement.

In conclusion, this 52-year-old male with congestive cardiomyopathy was maintained on intravenous milrinone for 17 days after the failure of catecholamine and intra-aortic balloon pump therapy. We recommend that milrinone should be considered as a bridge to transplantation when  $\beta$  agonist therapies have failed.

### References

1. JASKI BE, FIFER MA, WRIGHT RF, BRAUNWALD E, COLUCCI WS. Positive inotropic and vasodilator actions of milrinone in patients with severe congestive cardiac failure. Dose-response relationships and comparison to nitroprusside. *Journal of Clinical Investigation* 1985; **75**: 643–9.
2. MONRAD ES, BAIM DS, SMITH HS, LANOE A, BRAUNWALD E, GROSSMAN W. Effects of milrinone on coronary hemodynamics and myocardial energetics in patients with congestive heart failure. *Circulation* 1985; **71**: 972–9.
3. BENOTTI JR, LESKO LJ, MCCUE JE, ALPERT JS. Pharmacokinetics and pharmacodynamics of milrinone in chronic congestive heart failure. *American Journal of Cardiology* 1985; **56**: 685–9.
4. BROWN L, NABAUER M, ERDMANN E. The positive inotropic response to milrinone in isolated human and guinea pig myocardium. *Naunyn Schmiedeberg's Archives of Pharmacology* 1986; **334**: 196–201.
5. SUTKO JL, KENYON JL, REEVES JO. Effects of amrinone and milrinone on calcium influx into the myocardium. *Circulation* 1986; **73**: 52–8.

6. MOTULSKY HJ, INSEL PA. Adrenergic receptors in man. Direct identification, physiologic regulation, and clinical alterations. *New England Journal of Medicine* 1982; **307**: 18-29.
7. LEJEMTEL TH, MASKIN CS, MANCINI D, SINOWAY L, FELD H, CHADWICK B. Systemic and regional hemodynamic effects of captopril and milrinone administered alone, in patients with heart failure. *Circulation* 1985; **72**: 364-9.
8. CODY RJ, KUBO SH, COVIT AB, MULLER FB, RUTMAN H, LEONARD D, LARAGH JH, FELTSCHUH J, PREIBISZ J. Regional blood flow and neurohormonal responses to milrinone in congestive heart failure. *Clinical Pharmacology and Therapeutics* 1986; **39**: 128-35.
9. CINQUEGRANI M, MESNER C, BAGGS JG, FITZPATRICK PG, VAKIENER AR, LAING CS, BIDDLE TL, HOOD, WB. Effects of continuous milrinone on forearm and hepatic blood flows. *Clinical Research* 1984; **32**: 155A.



CASE REPORT

## Massive acute arsenic poisoning

D. M. JOLLIFFE, A. J. BUDD AND D. J. GWILT

### Summary

*A 28-year-old male ingested 75 g of arsenic trioxide in a successful suicide attempt. The presentation, management and postmortem findings are presented and discussed.*

### Key words

*Poisoning, acute; arsenic.*

### Case history

A 28-year-old self-employed analytical chemist attempted suicide by drinking a bottle of vodka and ingesting 75 g of arsenic trioxide. He fell asleep in his laboratory, awoke 4 hours later and admitted himself to hospital.

On admission he was vomiting profusely and had copious watery diarrhoea. He had some abdominal discomfort, but there was no evidence of gastrointestinal bleeding. On examination he was alert and cooperative, but shocked (blood pressure 85/60 mmHg, pulse 100 beats/minute). His chest was clear, and his abdomen soft with bowel sounds; he had absent knee jerks, but good limb power and flexor plantar responses.

Initial blood results showed that he was haemoconcentrated (haemoglobin 17.9 g/dlitre, haematocrit 0.57/litre) with normal urea, creatinine and electrolytes. Blood gases on air revealed a metabolic acidosis ( $P_{aO_2}$  12.9 kPa,  $P_{aCO_2}$  3.7 kPa, base deficit 11 mmol/litre). His chest X ray showed a radiopaque substance in the stomach.

Intravenous fluids were given, a urinary catheter inserted, and his stomach was washed out. Large volumes of saline were given via a nasogastric tube to purge the bowel. A suspension of activated charcoal was given to bind any unabsorbed arsenic. DMSA (2, 3-dimercaptosuccinic acid) capsules were brought by courier from the National Poisons Information Service and given orally. However, the patient vomited these and later dimercaprol was given intramuscularly.

The patient was transferred to the Intensive Care Unit after initial resuscitation. Over the next 11 hours he remained alert, but continued to lose large amounts of body fluids. His central venous pressure was maintained

between 0 and 9 cmH<sub>2</sub>O above the midaxillary line with 1 litre/hour of intravenous fluids (50% colloid, 50% crystalloid), with added potassium chloride (total 180 mmol). His urine output fell. A Swan Ganz catheter confirmed adequate left heart filling pressures (pulmonary artery wedge pressure 12 mmHg). Dopamine (at 2 µg/kg/minute) was given but the urine output failed to improve (27 ml/hour), and his creatinine rose to 304 µmol/litre. He became more acidotic and was given aliquots of 8.4% sodium bicarbonate solution. His temperature rose to 38.4°C per axilla.

The patient was transferred to a centre with dialysis facilities because of deteriorating renal function. He remained alert and cooperative during the transfer, which took an hour. He had stopped vomiting and had an unquenchable thirst. His systolic blood pressure fell and was only maintained between 65 and 75 mmHg by three pressurised infusion lines. His breathing became progressively more acidotic and he was unable to move his legs, although sensation remained intact.

Sixteen hours after ingestion, having had no previous arrhythmia, the patient developed a bradycardia and then asystole. He died despite attempted resuscitation.

At postmortem, the stomach contained 0.74 g of arsenic trioxide. The blood level of arsenic was 2.3 mg/litre (normal is less than 0.03 mg/litre). All organs showed congestion and the gastrointestinal mucosa was haemorrhagic. In the body of the stomach there were dilated thrombosed submucosal veins, and the lumen contained a mucosal cast due to sloughing of part of the gastric mucosa. The liver was grossly enlarged, soft, yellow and fatty. The myocardium was considerably congested with a normal coronary circulation.

D.M. Jolliffe, BSc, MRCP, DA, Senior House Officer, Department of Anaesthetics and Intensive Care, A.J. Budd, MB, FFARCS, Consultant Anaesthetist, D.J. Gwilt, MA, DM, FFARCS, MRCP, Consultant Physician, Milton Keynes General Hospital, Standing Way, Eaglestone, Milton Keynes, Buckinghamshire MK6 5LD.

Correspondence should be addressed to Dr Gwilt please.

Accepted 20 July 1990.

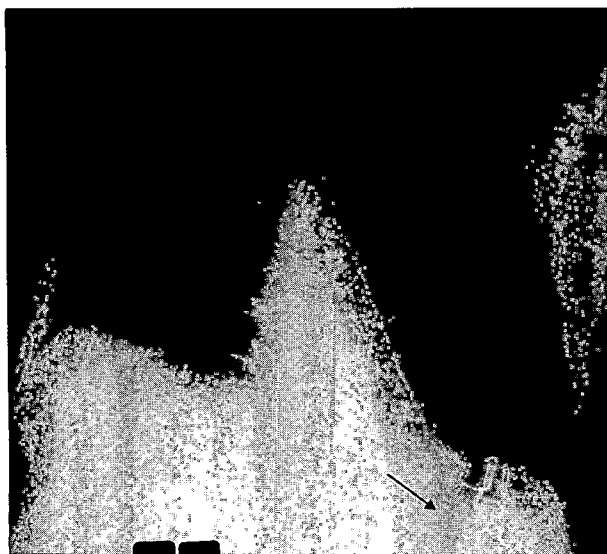


Fig. 1. Patient's chest X ray showing a radiopaque substance in the stomach (arrowed).

### Discussion

Arsenic (As) is a member of the nitrogen group (atomic weight 75) and is classified as a transitional element, a group known as heavy metals. It commonly forms complexes with metals and also readily reacts with carbon, oxygen and hydrogen forming covalent bonds.<sup>1</sup> It can exist in three different valency states: the metalloid (zero oxidation state), arsenite (trivalent), and arsenate (pentavalent). The toxicity of arsenic-containing compounds varies with the valency state (arsine,  $\text{AsH}_3$ , a gaseous hydride of  $\text{As}^{3+}$ , being the most toxic), the physical state of the compound, and the rate of absorption and elimination. Consequently, arsenic poisoning is not a single clinical entity.

Our patient took arsenic trioxide,  $\text{As}_2\text{O}_3$ . This is poorly water soluble and its toxicity greatly depends upon its physical state; it is less toxic when coarsely powdered, since it can be eliminated in the faeces before it dissolves. After absorption it is bound to the protein moiety of haemoglobin; it is cleared from the intravascular space within 24 hours and concentrated in the liver, kidneys, spleen, lungs and gastrointestinal tract. It is eliminated slowly by the kidneys as methylated arsenic. After 2 weeks any remaining arsenic is found in the hair, nails and skin and is slowly lost by shedding.

Trivalent arsenic inhibits sulphhydryl-containing enzymes by binding to the sulphhydryl groups. This causes widespread endothelial cellular toxicity resulting in capillary damage, generalised vasodilatation and transudation of plasma. There is massive transudation of fluid into the bowel lumen with mucosal vesicle formation and sloughing of tissue fragments. Symptoms of hypovolaemic shock occur, followed by acute tubular necrosis, convulsions, coma and death.<sup>2</sup>

If the patient survives the initial hypovolaemic phase, renal, hepatic and myocardial damage may occur. Neurological complications such as peripheral neuropathy and encephalopathy are also seen. Our patient exhibited most of these problems progressively over the 16-hour period.

Figures for the lethal dose of arsenic vary from author to author, but it is probably in the 200–300 mg range. Recovery from 10 g has occurred, but a dose of 20 mg may be life-threatening.<sup>1</sup> Our patient took 75 g and, in retrospect, it is likely that death was inevitable, particularly in view of the 4-hour delay before seeking medical help.

The principles of management are vigorous resuscitation and elimination of the poison. Fluid resuscitation must be aggressive. Invasive pressure monitoring, including left heart pressure monitoring, is advisable. However, death may still ensue from myocardial damage and peripheral circulatory collapse. We think we were able to replace the fluid loss in our patient; the right and left heart filling pressures were maintained at acceptable levels. However, the patient ultimately died of circulatory failure. It is likely that this was caused by myocardial failure, supported by the postmortem report of myocardial congestion. Consideration might perhaps be given to advanced forms of cardiac support (such as intra-aortic balloon pumping) where available.

Elimination of the poison depends upon purging the bowel and using chelating agents. We used large volumes of saline through a nasogastric tube to purge the bowel, and gave a suspension of activated charcoal to bind any unabsorbed arsenic.

Chelating agents act by reversing the binding of arsenic to sulphhydryl-containing enzymes, and the standard chelating agent for arsenic poisoning is dimercaprol (British anti-lewisite). However, it is toxic itself and causes nausea, vomiting, hypertension, tachycardia and a feeling of constriction in the throat and chest. Water-soluble analogues of dimercaprol, such as DMSA (2, 3-dimercaptosuccinic acid), are under investigation since they can be given orally and are less toxic.<sup>3</sup> However, our patient was vomiting so much that he could not tolerate oral DMSA and we later used dimercaprol without ill effect. Dimercaprol is likely to be available in all District General Hospitals, unlike DMSA which may have to be obtained from the Regional Poisons Unit. In a patient with massive acute arsenic poisoning, who is likely to be vomiting, it is better to use dimercaprol (which can be given parenterally and which will be immediately available) rather than wait to use DMSA which may not be absorbed.

Within the first 24 hours exchange transfusion may be of help in clearing the blood of arsenic.<sup>1</sup> Haemodialysis is recommended only in the presence of renal failure.<sup>4</sup> Functioning kidneys can excrete more than 100 mg of arsenic in 24 hours, whereas dialysis has been reported to remove about 4 mg over a 4-hour period.

Examination of the chest X ray showed radiopaque flecks in the stomach. We think this is arsenic, supported by finding arsenic in the stomach at postmortem. Radiopaque stomach contents on abdominal X ray in arsenic trioxide poisoning have been reported.<sup>5</sup> This radiological sign may be useful in identifying arsenic, or other heavy metal poisoning, when it has proved impossible to obtain an accurate history. It might also be a semiquantitative way of assessing the elimination of arsenic from the gastrointestinal tract.

In conclusion, we have presented a fatal case of massive acute arsenic poisoning. Aggressive fluid resuscitation is necessary, coupled with myocardial support. Elimination of the poison depends upon purging the bowel and the use of activated charcoal and an effective antidote. Based upon

## Potential errors in pulse oximetry

### III: Effects of interference, dyes, dyshaemoglobins and other pigments\*

A. C. RALSTON, R. K. WEBB AND W. B. RUNCIMAN

#### Summary

*Electrosurgery, patient motion and some types of lighting can cause errors in saturation readout; it is recommended that probes should be shielded from ambient lighting. Intravenous dyes can introduce gross but transient errors, which may also be present in in vitro measurements. Carboxyhaemoglobin causes overestimation of fractional saturation by an amount less than, but possibly close to, the percent of carboxyhaemoglobin present. Methaemoglobin causes the pulse oximeter readout to tend towards 85%. Fetal haemoglobin and bilirubin introduce no significant error, although they may interfere with in vitro measurements. Skin pigmentation can result in a slight decrease in accuracy. Nail polish may cause up to 6% underestimation of saturation; it is recommended that probes should be mounted sideways on fingers with nail polish or long nails. Adhesive tape or a vinyl glove across the probe has no demonstrable effect on accuracy. A blood sample should be analysed by a multiwavelength in vitro oximeter when an erroneous pulse oximeter reading is suspected, although errors may be introduced in the in vitro reading by fetal haemoglobin, bilirubin and intravenous dyes.*

#### Key words

*Equipment; pulse oximeter.  
Measurement.*

The performance and some potential errors of pulse oximeters under conditions of good and poor perfusion, and high and low saturation were reported before.<sup>1–5</sup> This paper is a review of other potential errors and the clinical implications will be reviewed later.<sup>6</sup>

#### Effects of interference

The pulse oximeter signal can be corrupted by interference from ambient light, electromagnetic signals and motion. Emergency Care Research Institute (ECRI) tested 13 models of pulse oximeter for the effects of interference by electrosurgical units, phototherapy and surgical lamps, and shaking and vibration.<sup>4</sup> These results are summarised below and compared with other reports where possible.

#### Electrosurgery

Electrosurgical interference caused erroneous readings in six of the 13 units with no clear warning that the signal was unsatisfactory; three were unaffected and the rest displayed a clear interference warning signal. Interference can be

reduced if the distance of the pulse oximeter probe from the surgical site is made as great as possible.

#### Motion

All of the units in the ECRI trial were affected to some degree by motion of the probe, such as heavy shaking, but the saturation values changed only minimally for simulated postoperative tremors most likely to be encountered clinically. Norley<sup>7</sup> inflated a blood pressure cuff to above systolic blood pressure on one subject and demonstrated that gentle shaking of the arm caused a pulse oximeter to display a saturation of 90%. He suggested that this may cast doubt on the validity of the use of pulse oximetry during cardiopulmonary resuscitation since movement artefact may cause false readings.

The susceptibility of a pulse oximeter to motion error is influenced by the duration of time over which the pulsing signals are averaged to give a final reading (usually 5–10 seconds), which may be changed by the user with some units. Longer averaging times reduce motion interference but increase the time delay between rapidly

A.C. Ralston, BSc, BAppSc, Medical Physicist, R.K. Webb, MB, BS, FFARACS, Senior Staff Specialist, W.B. Runciman, BSc(Med), MB, BCh, FFARACS, PhD, Professor and Head, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide SA 5000, Australia.

Correspondence should be addressed to Professor W.B. Runciman.

\*Combined study with Australian Patient Safety Foundation, GPO Box 400, Adelaide, South Australia 5001.

Accepted 6 August 1990.

changing saturations and the displayed reading. Correlating a pulse oximeter signal with an ECG waveform may help reject motion artefact, as can be done with Nellcor and Criticare pulse oximeters.

### Light

Three of the units in ECRI study were moderately affected by external light sources and one decreased from 97% to 93% for the entire exposure time without a warning signal. However, the rest were unaffected by the particular lights used in the test.

Pulse oximeters are designed to measure ambient light while the light emitting diodes (LEDs) in the probes are off, and then to subtract this background reading from the signal measured during light transmission through the finger to help prevent errors from light interference. Problems can occur when the high frequency modulation of the ambient light, which is tied to the mains frequency, is close but slightly different, from the LEDs on-off cycle, producing a pseudo-pulsatile waveform which is unrelated to the patient's own arterial pressure waveform.<sup>8</sup> Many manufacturers programme their oximeter LED on-off cycles to run at exactly the local mains frequency (50 or 60 Hz) to reduce this possibility.

Several clinical cases are reported in which pulse oximeters displayed high saturation values while the patient was obviously cyanosed,<sup>8-10</sup> or displayed full saturation and heart rate data, while the probe was actually off the patient.<sup>11,12</sup> Surgical, fluorescent and heating lamps are the common sources of this error. This problem is readily preventable by shielding the probe with dark cloth or other opaque material. Two groups have reported the use of aluminium foil packets from alcohol swabs to shield probes from external light sources.<sup>13,14</sup>

Some probes incorporate opaque side shielding, but most have open sides. We recommend the routine use of opaque cloth or rubber wrapped around the probe to reduce the ambient light reaching the light detector.

### Effects of intravenous dyes

Intravenous dyes such as methylene blue, indigo carmine, indocyanine green and fluorescein can cause erroneously low pulse oximeter saturation,  $\text{SpO}_2$ ,<sup>5</sup> readings with pulse oximetry due to peculiar light absorption characteristics.<sup>15,17</sup> These false readings are transitory and easily recognised if the user is aware of the potential for error. The effect of the dye is usually minimal within a minute of administration due to the dilution of the dye and its rapid distribution and/or clearance by the liver.<sup>18,19</sup>

Eisenkraft<sup>20</sup> points out that if there is doubt as to whether the decrease in  $\text{SpO}_2$  is caused by true hypoxia, a cross check measurement of either  $\text{SaO}_2$  or  $\%\text{HbO}_2$  by an *in vitro* oximeter will be affected by the same problem, and therefore cannot be used as an absolute reference. The size and sign of the error will vary according to dye and the wavelengths of the light used in the instrument.

Instrumentation Laboratory states that indocyanine green produces little interference in the range of wavelengths used by the IL 482 cooximeter (530–630 nm).<sup>21</sup> Methylene Blue and Evans Blue may erroneously increase the MetHb reading, but it is not specified by how much. However, oxygen tension measurements made with a blood gas

machine will not be affected by the presence of the dye, and constitute the obvious cross-check if there is any doubt.

There will still be a small effect on  $\text{SpO}_2$ , contrary to what Unger suggests,<sup>18</sup> when the dye concentration has reached equilibrium between the pulsatile and non-pulsatile tissue components at the probe site. The pulse oximeter cannot determine, in the presence of dye, what light absorbing substances are giving rise to the measured ratio of red to infrared intensities from which it estimates saturation. It will give an incorrect reading, although the error is likely to be so small as to be undetectable. Gabrielczyk and Buist<sup>22</sup> studied pulse oximeter accuracy in 21 postoperative patients, 11 of whom had intra-operative Patent Blue V dye infusions, and found no significant bias attributable to the dye.

Thus dyes can cause significant errors in oxygen saturation estimates by pulse oximeters due to interference with light absorption, but the effect is transient and easily recognised. Blood samples for *in vitro* analysis should not be drawn within a few minutes of the injection of dye because of interference by the same mechanism.

### Effects of dyshaemoglobin and other pigments

#### Carboxy- and methaemoglobin

Haemoglobin species such as carboxyhaemoglobin (HbCO) and methaemoglobin (MetHb) differentially absorb the red and infrared light from the pulse oximeter LEDs and thus affect the oxygen saturation reading.

Pulse oximeters are empirically calibrated using healthy volunteers with normal levels of HbCO and MetHb, usually less than 2%. Predictions of how much each haemoglobin species acts like  $\text{HbO}_2$  and how this will change the functional and fractional saturations, and claims that pulse oximeters actually detect reduced haemoglobin<sup>23</sup> can hinder understanding of the principle of pulse oximetry. A pulse oximeter simply measures the ratio of the transmitted intensities of the two wavelengths due to all pulsatile absorbing material, then uses this ratio to find the equivalent oxygen saturation in a 'look-up' table constructed from data for 'normal' individuals. MetHb and HbCO affect this ratio and cause erroneous  $\text{SpO}_2$  readings using the wavelengths of 660 nm and 940 nm. Oximeters using other wavelengths will be affected differently.

HbCO has the greater effect, causing overestimation of oxygen saturation by an amount less than the HbCO percentage. Davies<sup>24</sup> states without reference that 10% HbCO causes overestimation of  $\text{SpO}_2$  by 2–3% for saturations greater than 85%. From studies on dogs, Barker and Tremper<sup>25</sup> found the amount of  $\text{SpO}_2$  overestimation to be closer to the HbCO percentage for HbCO from 0–80%. Henderson<sup>26</sup> found to the contrary: in a study of preterm neonates there was no significant correlation between HbCO levels (3.2–9.9%, average 7.2%) and the difference between  $\text{SpO}_2$  and IL 282 cooximeter  $\%\text{HbO}_2$  measurements.

It is a common misconception<sup>23,27</sup> that HbCO is 'seen' by the pulse oximeter as  $\text{HbO}_2$ , and that the  $\text{SpO}_2$  reading is the sum of  $\%\text{HbO}_2$  and  $\%\text{HbCO}$ . This would only be possible if HbCO had the same absorption coefficients as  $\text{HbCO}_2$  at 660 nm and 940 nm, which is not so.

MetHb has approximately the same absorption coefficient at both wavelengths, and if enough MetHb is present

to dominate all pulsatile absorption the pulse oximeter will measure a red to infrared absorption ratio of about 1:1. This corresponds in most pulse oximeter 'look-up' tables for normal blood to an oxygen saturation of 85%.<sup>27</sup> The presence of MetHb will thus bias the  $SpO_2$  reading towards 85%, which will result in over- or underestimation of saturation for %HbO<sub>2</sub> values below and above 85% respectively. Clearly then, a high MetHb concentration can mask profound desaturation, as reported by several groups for MetHb concentrations ranging from 13% to 64%.<sup>28-30</sup> A striking example is the case of a neonate with an actual %HbO<sub>2</sub> of 45%, MetHb% of 26% and a  $SpO_2$  of 85%.<sup>30</sup> Low levels of MetHb decrease the pulse oximeter saturation reading by about half the MetHb%.<sup>23</sup>

The IL 482 manual<sup>21</sup> states that the spectral absorption of sulphhaemoglobin is similar to that of methaemoglobin in its operating range (530–630 nm) and will be measured as such, thus its detection is not possible by the use of two or four wavelength oximetry. We have found no reference in the literature to the effect of sulphhaemoglobin on pulse oximeter readings.

Significant errors in pulse oximeter readings can thus be caused by the presence of dysaemoglobins. HbCO will cause an erroneous increase in  $SpO_2$  by an amount less than, but possibly close to, the percentage of HbCO. Large quantities of MetHb (> 10%) can result in a stable pulse oximeter reading of 85% regardless of actual  $SpO_2$ . Low quantities of MetHb will reduce a high  $SpO_2$  reading by about half the MetHb%.

#### Fetal haemoglobin

Newborn full term infants can have up to 75% of total haemoglobin in the form of fetal haemoglobin.<sup>31</sup> Mendelson and Kent<sup>32</sup> recently showed that fetal haemoglobin has almost identical absorption to adult haemoglobin for all light wavelengths from 650 nm to 1000 nm, which is the range in which all pulse oximeters operate. Thus pulse oximeters should be as accurate in the presence of fetal haemoglobin as with adult haemoglobin alone. This theory has been verified by many clinical trials, a review of which is in Dziedzic and Vidyasagar's article on pulse oximetry in neonatal intensive care.<sup>33</sup>

One complication is that fetal haemoglobin may cause overestimation of HbCO when measured by a cooximeter operating with wavelengths below 650 nm,<sup>34,35</sup> as the fetal and adult absorption characteristics differ in this region. Instrumentation Laboratory provide an algorithm to compensate for the effect of fetal haemoglobin on the measurement of HbO<sub>2</sub> and HbCO.<sup>21</sup> A correction value proportional to HbO<sub>2</sub> and the fetal haemoglobin percentage is added to the measured HbO<sub>2</sub> and subtracted from the measured HbCO. The uncorrected IL 482 readings will underestimate HbO<sub>2</sub> and overestimate HbCO by approximately 1% for 8% fetal haemoglobin, and by approximately 5.5% for 80% fetal haemoglobin in well oxygenated infants. The IL 482 corrects the readings automatically if the fetal haemoglobin is entered.

The presence of fetal haemoglobin has no significant effect on the  $SpO_2$  reading of a pulse oximeter, but will increase the HbCO and decrease the %HbO<sub>2</sub> measurements made with a cooximeter. If this is not accounted for it will appear that the discrepancy between %HbO<sub>2</sub> and  $SpO_2$  measurements is due to the presence of HbCO. This should

be borne in mind when assessing the oxygenation status of infants of up to one year in age.

#### Bilirubin

Taylor and Whitwam<sup>36</sup> state that a raised bilirubin concentration causes underestimation of oxygen saturation by pulse oximetry, but give no reference to support this. Conversely, three groups<sup>37-39</sup> could not show any effect on the accuracy of  $SpO_2$  readings with bilirubin levels up to 84.3 mg/dlitre. The absorption spectrum of bilirubin has a broad peak at 460 nm and two much smaller peaks at 560 nm and 600 nm.<sup>40</sup> Thus, bilirubin is unlikely to have any detectable effect on the absorption of the 660 nm and 940 nm wavelengths used by pulse oximeters.

High bilirubin levels may have an effect on absorption at the lower wavelengths used by cooximeters. Instrumentation Laboratory states that a bilirubin concentration of 20 mg/dlitre will cause less than 1% error in the measurement of the four main haemoglobin species. Clerbaux *et al.*<sup>41</sup> have proposed an equation for the correction of the Corning cooximeter measurements (Corning CO 2500, Medfield, MA) for bilirubin.

Thus the presence of bilirubin in the arterial blood will not introduce any significant errors in pulse oximetry measurements, but may cause a discrepancy between pulse oximeter and cooximeter readings.

#### Effects of pigments

Skin pigmentation, coloured disinfectants and other surface light absorbers should, in theory, not cause errors in  $SpO_2$  readings since the pigments absorb a constant fraction of the incident light, and the pulse oximeter uses only pulsatile absorption data. This is achieved by subtraction of the constant, or DC, component of the transmitted light from the total transmitted light, leaving the pulsatile, or AC, component. The height of the AC component is reduced by nonpulsatile absorption during transmission. This is compensated for by division of the AC component of the transmitted intensity by the DC component of the transmitted intensity to give the corrected AC signal. This parameter is sometimes called the pulse-added absorbance.<sup>42</sup>

#### Skin pigmentation

Cecil *et al.* assessed the accuracy of two pulse oximeters on 152 patients (15 black), and showed an apparent greater inaccuracy in  $SpO_2$  readings for black patients, and suggested that this could be due to increased LED power output.<sup>43</sup> Ries *et al.*<sup>44</sup> studied skin colour and ear oximetry of 187 patients with a range of skin colours and concluded that there were significantly more signal quality problems and slightly decreased accuracy in black patients.

Conversely, Gabrielczyk and Buist<sup>22</sup> found no significant bias attributable to pigmented skin in a study of 21 patients (four black), but the population size was probably too small to show up minor differences in pulse oximeter performance.

### Nail varnish

Similarly conflicting data exist for the effect of nail polish on saturation readings. In theory there should be no effect since the absorbance of light by varnish is nonpulsatile and is cancelled out of the saturation calculation, as is tissue absorption.

Kataria and Lampkins<sup>45</sup> applied nail polish (colour unspecified) to the nails of 15 volunteers and found no effect on  $\text{SpO}_2$  readings. Cote *et al.*<sup>46</sup> applied a variety of coloured nail polishes to the nails of 14 subjects. They found that black, blue and green, but not red or purple, nail varnish caused a significant lowering of  $\text{SpO}_2$  readings of the Nellcor N-100. The average errors were 3%, 5% and 6% for the black, green and blue respectively. They attribute the difference in effect to the absorption spectra of the five colours, but do not suggest why this constant absorption should affect the pulse oximeter calculation of oxygen saturation. The N-100 increases its light output in response to low detected light levels so it is possible that the higher LED current caused shift in the output spectrum,<sup>23</sup> which altered the measured red-infrared ratio and the saturation reading.

The reports that neither the finger of a vinyl glove<sup>47</sup> nor adhesive tape<sup>48</sup> between the finger and the oximeter probe caused consistent differences from readings on bare fingers are more in line with theory.

Severinghaus suggests mounting the probe side-to-side on the finger.<sup>23</sup> White and Boyle<sup>49</sup> used 10 volunteers with and without nail polish to check this strategy with two pulse oximeters, and found that both disposable and nondisposable probes could be mounted sideways on the finger and gave the same readings as uncovered nails. This technique may also help to avoid the problem of saturation underestimation caused by only partial placement of the light source over the finger because of very long fingernails, as reported by Tweedie.<sup>50</sup>

Pigmented skin and painted fingernails may reduce the accuracy of pulse oximetry. Skin pigmentation has a small effect, only seen as a slight decrease in accuracy over a large number of samples. Blue nail polish was found to have the greatest effect, causing an average underestimation of saturation of 6%. We recommend that probes be mounted sideways on the finger if nail polish is present.

### Conclusions

A hazard in the use of pulse oximeters is a false reading under apparently ideal conditions. If the error is unrecognised it can lead to inappropriate medical action or ignorance of a situation which requires action. Lighting is probably the most insidious factor, since the false reading may be stable for long periods and have an apparently high signal quality. The errors introduced by dyes may be anticipated and are easily recognised by their transient nature. *In vitro* oximeters can be susceptible to the same errors so no direct measure of saturation can be made until the dye has cleared from the blood.

The presence of a high percentage of carboxyhaemoglobin or methaemoglobin can cause gross overreading of saturation and mask serious hypoxia. There is no failsafe method of guarding against this other than awareness of this limitation of pulse oximetry and by the repeated use of comparative baselines for  $\text{SpO}_2$ ,  $\%\text{SaO}_2$ , and  $\text{PaO}_2$ . Blood

samples should be analysed in a multiwavelength *in vitro* oximeter, which can measure  $\%\text{MetHb}$  and  $\%\text{HbCO}$ . Note that the  $\text{PO}_2$  may still be high, so the saturation value calculated by a blood gas machine may be erroneous.

The best defence against all these potential problems is a high index of suspicion and insight into the mechanism and potential significance of each of these problems. If a saturation reading is in doubt a limited test is for the staff member to place the pulse oximeter probe on his (her) own finger as near as possible to the original patient site to check that the reading is 97%, or as expected. When the  $\text{SpO}_2$  is suspected to be incorrect we recommend an initial analysis by a multi-wavelength *in vitro* oximeter. Now that pulse oximetry is in widespread use, it is suggested that *in vitro* saturation measurements by multi-wavelength oximeters should be routinely available in large centres.

### References

1. WEBB RK, RALSTON AC, RUNCIMAN WB. Potential errors in pulse oximetry. Part II: Effects of changes in saturation and signal quality. *Anaesthesia* 1991; **46**: 207–212.
2. CLAYTON DG, WEBB RK, RALSTON AC, DUTHIE D, RUNCIMAN WB. A comparison of the performance of twenty pulse oximeters under conditions of poor perfusion. *Anaesthesia* 1991; **46**: 3–10.
3. CLAYTON D, WEBB RK, RALSTON AC, DUTHIE D, RUNCIMAN WB. Pulse oximeter probes. A comparison between finger, nose, ear and forehead under conditions of poor perfusion. *Anaesthesia* 1991; **46**: 260–265.
4. EMERGENCY CARE RESEARCH INSTITUTE. Pulse oximeters. *Health Devices* 1989; **18**: 185–230.
5. RALSTON AC, WEBB RK, RUNCIMAN WB. Potential errors in pulse oximetry. Part I: Pulse oximeter evaluation. *Anaesthesia* 1991; **46**: 202–206.
6. RUNCIMAN WB, WEBB RK, RALSTON AC. Potential errors in pulse oximetry. Part IV: Clinical significance. (in preparation).
7. NORLEY I. Erroneous actuation of the pulse oximeter. *Anaesthesia* 1987; **42**: 1116.
8. COSTARINO AT, DAVIS DA, KEON TP. Falsely normal saturation reading with the pulse oximeter. *Anesthesiology* 1987; **67**: 830–1.
9. EMERGENCY CARE RESEARCH INSTITUTE. Pulse oximeter interference from surgical lighting. *Health Devices* February 1987: 50–1.
10. EMERGENCY CARE RESEARCH INSTITUTE. Ambient light interference with pulse oximeters. *Health Devices* Sept–Oct 1987: 346–7.
11. HANOWELL L, EISELE JH, DOWNS D. Ambient light affects pulse oximeters. *Anesthesiology* 1987; **67**: 864–5.
12. MUNLEY AJ, SIK MJ. An unpredictable and possibly dangerous artefact affecting a pulse oximeter. *Anaesthesia* 1988; **43**: 334.
13. ZABLOCKI AD, RASCH DK. A simple method to prevent interference with pulse oximetry by infrared heating lamps. *Anesthesia and Analgesia* 1987; **66**: 913.
14. SIEGEL MN, GRAVENSTEIN N. Preventing ambient light from affecting pulse oximetry. *Anesthesiology* 1987; **67**: 280.
15. BARKER SJ, TREMPER KK. Pulse oximetry: applications and limitations. *International Anesthesiological Clinics* 1987; **25**: 155–75.
16. GORMAN ES, SHNIDER MR. Effect of methylene blue on the absorbance of solutions of haemoglobin. *British Journal of Anaesthesia* 1988; **60**: 439–44.
17. SCHELLER MS, UNGER RJ, KELNER MJ. Effects of intravenously administered dyes on pulse oximetry readings. *Anesthesiology* 1986; **65**: 550–2.
18. UNGER R, SCHELLER MS. More on dyes and pulse oximeters. *Anesthesiology* 1987; **67**: 148–9.
19. EIDE TR, HUMAYUN-SCOTT B, POPPERS PJ. More on dyes and pulse oximeters—Reply. *Anesthesiology* 1987; **67**: 149.
20. EISENKRAFT JB. Methylene blue and pulse oximetry readings: spuriouser and spuriouser! *Anesthesiology* 1988; **68**: 171.



21. INSTRUMENTATION LABORATORIES IL 482 Operator's Manual, Lexington, MA.
22. GABRIELCZYK MR, BUIST RJ. Pulse oximetry and postoperative hypothermia. *Anaesthesia* 1988; **43**: 402-4.
23. SEVERINGHAUS JW. Pulse oximetry uses and limitations. ASA 40th Annual Refresher Course Lectures and Clinical Update Program. American Society of Anesthesiologists Inc. 1989.
24. DAVIES M. Pulse oximetry. Australian Anaesthesia Lectures 1988: 149-55.
25. BARKER SJ, TREMPER KK. The effect of carbon monoxide inhalation on pulse oximetry and transcutaneous pO<sub>2</sub>. *Anesthesiology* 1987; **66**: 677-9.
26. HENDERSON GW. Accuracy and reliability of pulse oximetry in premature neonates with respiratory distress. *Journal of the American Association of Nurse Anesthetists* 1988; **56**: 224-8.
27. ALEXANDER CM, TELLER LE, GROSS JB. Principles of pulse oximetry: theoretical and practical considerations. *Anesthesia and Analgesia* 1989; **68**: 368-76.
28. ANDERSON ST, HAJDUCZEK J, BARKER SJ. Benzocaine-induced methemoglobinemia in adults: accuracy of pulse oximetry with methemoglobinemia. *Anesthesia and Analgesia* 1988; **67**: 1099-1101.
29. RIEDER HU, FREI FJ, ZBINDEN AM, THOMSON DA. Pulse oximetry in methaemoglobinaemia. *Anaesthesia* 1989; **44**: 326-7.
30. WATCHA MF, CONNOR MT, HING AV. Pulse oximetry in methemoglobinemia. *Am. J. Dis. Child.* 1989; **143**: 845-7.
31. OSKI FA. Clinical implications of oxyhemoglobin curve in the neonatal period. *Critical Care Medicine* 1979; **7**: 412-18.
32. MENDELSON Y, KENT JC. Variations in optical absorption spectra of adult and fetal hemoglobins and its effect on pulse oximetry. *IEEE Transactions on Biomedical Engineering* 1989; **36**: 844-8.
33. DZIEDZIC K, VIDYASAGAR D. Pulse oximetry in neonatal intensive care. *Clinics in Perinatology* 1989; **16**: 177-97.
34. ZWART A, BUURSMA A, OESEBURG B, ZIJLSTRA WG. Determination of hemoglobin derivatives with the IL 282 CO-oximeter as compared with a manual spectrophotometric five wavelength method. *Clinical Chemistry* 1981; **27**: 1903-7.
35. ZIJLSTRA WG, BUURSMA A, KOEK J, ZWART A. Letter to the Editors. *Clinical Chemistry* 1983; **29**: 1556.
36. TAYLOR MB, WHITWAM JG. The current status of pulse oximetry. *Anaesthesia* 1986; **41**: 943-9.
37. VEYCKEMANS F, BAELE P, GUILLAUME JE, WILLEMS E, ROBERT A, CLERBAUX T. Hyperbilirubinemia does not interfere with hemoglobin saturation measured by pulse oximetry. *Anesthesiology* 1989; **70**: 118-22.
38. RAMANATHAN R, DURAD M, LARRAZABAL C. Pulse oximetry in very low birth weight infants with acute and chronic lung disease. *Pediatrics* 1987; **79**: 612-17.
39. PRAUD JP, CAROFILIS A, BRIDEY F, LACAILE F, DEHAN M, GAULTIER CL. Accuracy of two wavelength pulse oximetry in neonates and infants. *Pediatric Pulmonology* 1989; **6**: 180-82.
40. AMAZON K, SOLONI F, RYWLIN AM. Separation of bilirubin from hemoglobin by recording derivative spectrophotometry. *American Journal of Clinical Pathology*. 1981; **75**: 519-23.
41. CLERBAUX T, WILLEMS E, NULLENS W, BAELE PL, VEYCKEMANS F, HASSOUN A. Interférence de la bilirubine sur la saturation oxyhémoglobinée mesurée par voie photométrique. *Nouvelle Presse Médicale* 1988; **17**: 960.
42. TREMPER KK, BARKER SJ. Pulse oximetry. *Anesthesiology* 1989; **70**: 98-108.
43. CECIL WT, THORPE KJ, FIBUCH EE, TUOHY GF. A clinical evaluation of the accuracy of the Nellcor N-100 and the Ohmeda 3700 pulse oximeters. *Journal of Clinical Monitoring* 1988; **4**: 31-6.
44. RIES AL, PREWITT LM, JOHNSON JJ. Skin color and ear oximetry. *Chest* 1989; **96**: 287-90.
45. KATARIA BK, LAMPKINS R. Nail polish does not affect pulse oximeter saturation. *Anesthesia and Analgesia* 1986; **65**: 824.
46. COTE CJ, GOLDSTEIN EA, FUCHSMAN WH, HOAGLIN DC. The effect of nail polish on pulse oximetry. *Anesthesia and Analgesia* 1988; **67**: 683-6.
47. ACKERMAN WE, JUNEJA MM, BAUMANN RC, KACZOROWSKI DM. The use of a vinyl glove does not affect pulse oximeter monitoring. *Anesthesiology* 1989; **70**: 558-9.
48. READ MS. Effect of transparent adhesive tape on pulse oximetry. *Anesthesia and Analgesia* 1989; **68**: 701-2.
49. WHITE PF, BOYLE WA. Nail polish and oximetry. *Anesthesia and Analgesia* 1989; **68**: 546-7.
50. TWEEDIE IE. Pulse oximeters and finger nails. *Anaesthesia* 1989; **44**: 268.

## Assessment of a hygroscopic heat and moisture exchanger for paediatric use

K. A. WILKINSON, A. CRANSTON, D. J. HATCH AND M. E. FLETCHER

### Summary

*A laboratory study of a widely available heat and moisture exchanger marketed for paediatric use was undertaken. The deadspace, measured by volume displacement, was 12 ml, similar to that of a standard catheter mount for paediatric use. Pressure drop across the device was measured at several different flows in five samples of the device in both the dry and wet state. Calculated resistance proved to be markedly lower when compared with that of other anaesthetic equipment such as tracheal tubes, and with similar humidification devices for paediatric use.*

### Key words

*Equipment; humidifiers, deadspace.*

*Airway; resistance.*

Heat and Moisture Exchangers (HMEs) are now used widely in adult anaesthesia and intensive care.<sup>1–4</sup> However, their use in the paediatric patient has been restricted through concerns about deadspace (which may lead to rebreathing, particularly in the infant) internal resistance (which may result in increased work of breathing) and limited efficiency (with danger of inspissated secretions leading to blockage of the tracheal tube).

These concerns are not based on firmly established data. We therefore measured deadspace and resistance *in vitro* in a widely available HME marketed for use in children. Resistance (R) seemed likely to increase when the device is wet; consequently, we measured pressure drop across the HME before and after a 2-hour wetting period.

### Materials and methods

The dimensions of five sample Portex Thermovent 600 HMEs were measured. A Sartorius 1201 MP2 balance was used to weigh these HMEs before and after the wetting period. Deadspace was measured using a water displacement method. The 22-mm female end of five of the HMEs was sealed, and each unit was filled carefully with distilled water using graduated syringes, making sure no air bubbles

remained. Each device was then tapped to remove excess water and the procedure repeated.

Differential pressure gradient across the HMEs was measured using a 19-gauge metal needle connected to an industrial draught gauge (Combustion Instruments Ltd, model S706/A; accuracy to 0.01 cmH<sub>2</sub>O, 0.98 Pa) (Fig. 1). Pressures were measured at various air flows between 0.03 and 0.5 litres/second delivered from an accurate flowmeter (Rotameter Manufacturing Co, series 1100; accuracy  $\pm 2\%$ ). The procedure was repeated after wetting the devices.

Statistical analysis comprised the calculation of confidence intervals for pressure drop at each of the seven flows studied, using the measurements for the dry and the wet state. The wet HME rig (Fig. 2) consisted of a modified breathing system attached to a ventilator capable of delivering a small tidal volume,<sup>5</sup> a model lung, a Bennett cascade water bath adjusted to give a temperature of 35°C at the connexion with the model lung (Ohmeda Ltd, resistance 0.49 kPa/litre/second, compliance 0.5 ml/Pa), and the Thermovent 600. The ventilator was set to deliver a tidal volume of 150 ml at a rate of 25 cycles/minute, giving a peak inspiratory pressure of 1 kPa (10 cmH<sub>2</sub>O). The I:E ratio was 1:1.

K.A. Wilkinson,\* MB, BS, FCAnaes, DCH, MRCP, A. Cranston,† MB, BS, FCAnaes, Registrars, D.J. Hatch, MB, BS, FCAnaes, Consultant, Department of Anaesthesia, M.E. Fletcher,‡ BSc, SRN, RSCN, Research Sister, Respiratory Unit, The Hospital for Sick Children, Great Ormond Street, London WC1N 3JH.

\*Present appointment: Senior Registrar, Department of Anaesthesia, The London Hospital, Whitechapel, London, E1. Present address: Paediatric Intensive Care Unit, Royal Children's Hospital, Parkville, Melbourne, Victoria 3052, Australia.

†Present appointment: Senior Registrar, Department of Anaesthesia, New Cross Hospital, Wolverhampton, West Midlands.

Correspondence should be addressed to Dr D. Hatch please.

Accepted 3 August 1990.

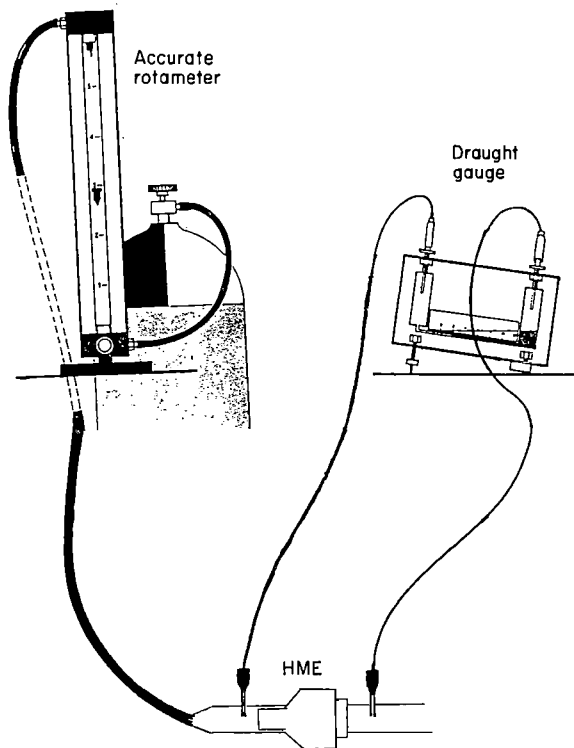


Fig. 1. Equipment to measure pressure drop (modified with permission, from Hatch).<sup>14</sup>

### Results

The external dimensions of the HMEs were 59 mm (length) × 29 mm (maximal diameter), with 15/22 mm connexions at either end. The mean deadspace of the five HMEs studied was 12 ml (range 11.7–12.3). There was a mean weight increase of 2.9 g after a 2-hour wetting period (Table 1). HME 5 showed a significantly smaller increase in weight than the other four but this was not reflected in subsequent resistance measurements.

There was a curvilinear increase in the pressure drop measured across the device with increasing flow. This proved to be greater when the device was wet at all flows except 4 litres/minute, (Table 2; Fig. 3). No significant change in resistance occurred at any flow rate, when the HMEs were wet.

Table 1. Dry and wet weights (g) of five HMEs (atmospheric pressure 1.0119 bar, temperature 23.4°C).

HME	Dry	Wet	Net increase
1	10.4078	13.776	3.368
2	10.532	13.275	2.743
3	10.508	13.526	3.018
4	10.548	13.964	3.416
5	10.457	12.411	1.954
Mean weight increase (95% confidence interval, 2.161–3.640)			2.899 g

### Discussion

Concerns about deadspace and resistance of HMEs have restricted their use in young children.<sup>6</sup> The results of this study have shown that some of these concerns are unfounded.

The measured deadspace of 12 ml was comparable with that quoted by the manufacturer<sup>7</sup> (11.3 ml) measured by a compressible volume technique. We recognise the limitations of deadspace measurement using a water displacement method when the device contains a hygroscopic element. However, subsequent deadspace measurements proved to be repeatable after the HME had been wetted. The effects of adding a 12-ml deadspace to the breathing system during spontaneous ventilation obviously depend on the size of the patient and any mandatory apparatus deadspace already incurred.<sup>8</sup>

However, because of gas mixing within apparatus, the actual effect of adding a deadspace to a breathing system *in vivo* may prove to be less than that predicted *in vitro*. Moreover, the deadspace of this HME comprises very little more than that imposed by the use of a standard catheter mount. The use of preformed tracheal tubes can reduce the total apparatus deadspace significantly by avoiding the use of a catheter mount.

The wet HME rig differed somewhat from that used in HEI 166 and in the draft BSI 88/528382.<sup>9,10</sup> Our system was comparable with that used by Buckley when assessing increases of resistance of in-line breathing filters.<sup>11</sup> Our aim was not to assess HME efficiency, but we appreciate the concerns expressed by Bethune<sup>12</sup> and others that systems which do not deliver fully saturated expired gases to the HME are probably not comparable to the clinical situation.

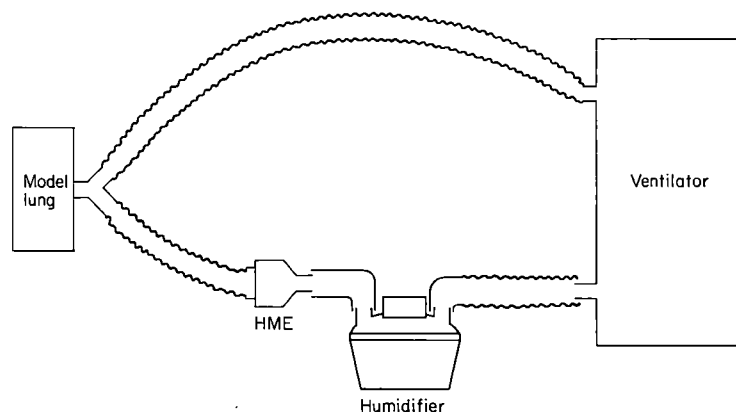


Fig. 2. Wet humidifier rig showing position of heat and moisture exchanger.

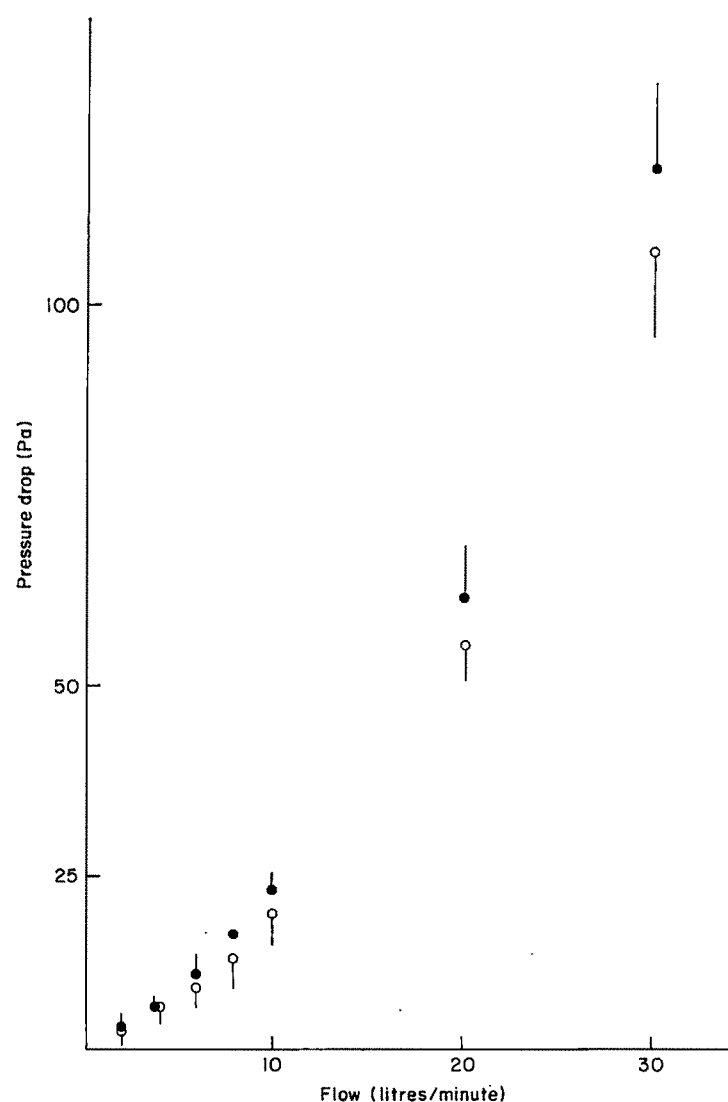


Fig. 3. Pressure drop across the Thermovent 600 HME. ○, dry; ●, wet; bars represent 1 SD.

We confirmed adequate wetting of our device by weighing before and after a 2-hour period in the system.

Table 2. Mean pressure drop with 95% confidence intervals (CI), and mean resistance, across the HMEs at various flows.

Flow (litres/minute)	Pressure drop (Pa)		Resistance (Pa/litre/second)
	mean	95% CI	mean
<i>Dry</i>			
2	2.5	1.2-3.7	81.6
4	6.1	4.1-8.1	101.6
6	8.2	6.3-10.2	82.3
8	12.7	8.8-16.7	98.0
10	17.8	14.6-21.3	111.5
20	53.8	48.9-58.7	163.0
30	106.8	94.6-119.0	213.6
<i>Wet</i>			
2	3.1	1.7-4.6	104.5
4	5.7	4.8-6.6	94.7
6	10.4	8.5-12.3	103.8
8	15.3	13.9-16.6	117.6
10	21.2	19.6-22.8	132.3
20	60.4	53.1-67.6	182.9
30	117.2	104.7-129.6	234.4

We found the HME to be heavily laden with excess water at the end of this time. We felt that this situation was unlikely to occur in a clinical setting and we therefore gently tapped the devices free of gross accumulated water before weighing. It is not recommended that HMEs are used in conjunction with water bath humidifiers but we chose to use this rig to ensure good HME saturation. A mean weight increase of 3 g suggested saturation of the HME element.<sup>13</sup>

We include a graphical representation of pressure drop across the HME (dry and wet) against flow (Fig. 3). The increase in resistance when the device was wet, as previously noted, is not statistically significant ( $p > 0.05$ ). Moreover, we feel that this increase is unlikely to be clinically important. Comparisons with resistance of size 3.5 mm internal diameter tracheal tubes without connector (686 Pa/litres/second)<sup>14</sup> and with pressure drop across an HME studied and recommended for neonatal use (80 Pa)<sup>15</sup> further emphasise the relatively small resistive load imposed by the Thermovent (pressure drop 9 Pa) at a flow of 5 litres/minute (Fig. 4).

The addition of a significant resistive load is important in terms of work of breathing.<sup>16</sup> We have demonstrated that,

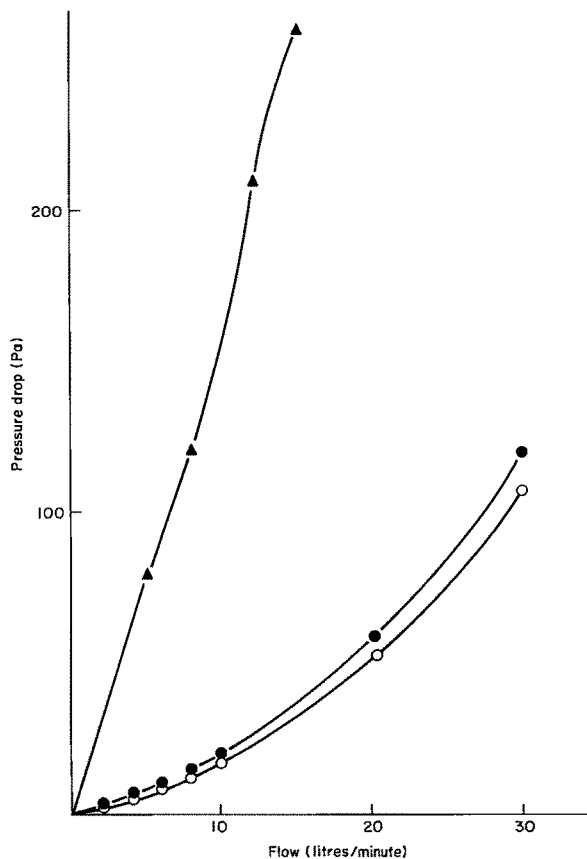


Fig. 4. Pressure drop across the Thermovent 600 compared with a neonatal HME. (Pressure drop for Cori device from reference 15, and from personal communication with the authors). ▲, Cori neonatal HME; ○, Thermovent 600 (dry); ●, Thermovent 600 (wet).

in the case of the Portex Thermovent under study, resistive load would appear to be acceptably low, both in comparison to standard anaesthetic apparatus such as tracheal tubes, and also to similar devices recommended for use in infants and children.<sup>4,15</sup>

#### Acknowledgments

The Thermovent 600 HMEs were supplied by the manufacturer (Portex Ltd, Hythe, Kent). We acknowledge the help

of Dr A. Gedeon for numeric clarification of data from reference 15.

#### References

1. WALKER AKY, BETHUNE DW. A comparative study of condenser humidifiers. *Anaesthesia* 1976; **31**: 1086-93.
2. STEWARD DJ. A disposable condenser humidifier for use during anaesthesia. *Canadian Anaesthetists' Society Journal* 1976; **23**: 191-5.
3. OH TE, THOMPSON WR, HAYWARD DR. Disposable condenser humidifiers in intensive care. *Anaesthesia and Intensive Care* 1981; **9**: 331-5.
4. GEDEON A, MEBIUS C. The hygroscopic condenser humidifier. A new device for general use in anaesthesia and intensive care. *Anaesthesia* 1979; **34**: 1043-7.
5. CHAKRABARTI MK, WHITWAM JG. A new valveless all-purpose ventilator. Description and laboratory evaluation. *British Journal of Anaesthesia* 1983; **55**: 1005-15.
6. HAY R, MILLER WC. Efficacy of a new hygroscopic condenser humidifier. *Critical Care Medicine* 1982; **10**: 49-51.
7. Portex technical advice on Thermovent heat and moisture exchangers. Portex Ltd, Hythe, Kent 1988.
8. CHARLTON AJ, LINDAHL SGE, HATCH DJ. Ventilatory responses of children to changes in deadspace volume. Studies using the T-piece (Mapleson F) system. *British Journal of Anaesthesia* 1985; **57**: 562-8.
9. British Standards Institution. Draft British Standard specification for heat and moisture exchangers for use in humidifying respired gases. BSI 1988, Document number 88/52832.
10. Health Equipment Information 166. Evaluation of Heat and Moisture Exchangers. DHSS 1987.
11. BUCKLEY PM. Increase in resistance of in-line breathing filters in humidified air. *British Journal of Anaesthesia* 1984; **56**: 637-43.
12. BETHUNE DW. Health equipment (dis)Information: HEI 166. evaluation of heat and moisture exchangers. *Anaesthesia* 1989; **44**: 161.
13. DUNCAN A. Use of disposable condenser humidifiers in children. *Anaesthesia and Intensive Care* 1985; **13**: 330.
14. HATCH DJ. Tracheal tubes and connectors used in neonates—dimensions and resistance to breathing. *British Journal of Anaesthesia* 1978; **50**: 959-64.
15. GEDEON A, MEBIUS C, PALMER K. Neonatal hygroscopic condenser humidifier. *Critical Care Medicine* 1987; **15**: 51-4.
16. LINDAHL SGE, CHARLTON AJ, HATCH DJ, PHYTHYON JM. Ventilatory responses to inspiratory mechanical loads in spontaneously breathing children during halothane anaesthesia. *Acta Anaesthesiologica Scandinavica* 1986; **30**: 122-7.

## Francis Percival de Caux (1892–1965)

An anaesthetist at odds with social convention and the law

D. J. WILKINSON

### Summary

*All doctors practice medicine within the confines of what is termed 'acceptable practice'. This acceptable practice is delineated by medical ethics, the actions of one's colleagues, social custom, and the laws of the country. Failure to conform to any or all of these constraints may result in professional ostracism or even loss of liberty. The life and work of Frances Percival de Caux clearly shows these effects in their most damaging manner.*

### Key words

History; F. P. de Caux.

F. P. de Caux qualified in medicine in 1921 from St Bartholomew's Hospital and became a prominent anaesthetist in the next two decades. He was an early protagonist of change in the laws relating to abortion, but unwisely broke those same laws and was sent to jail. After the second world war he became an early exponent of alternative medical therapy. His life story clearly shows the social changes that have occurred over the past 60 years and how these are reflected in medical practice and the laws of this country.

### Family background

Francis Percival de Caux was born with the surname Cowx in Takaka, New Zealand on the 26 November 1892. His father, Howard Percival Cowx, was an evangelist preacher who had been ordained in the diocese of Nelson in New Zealand in 1890 having emigrated from near Tamworth in Warwickshire. Francis was the eldest of four children, who were all subjected to a very strict religious upbringing. In a letter written in 1957 he referred to his home life as being 'morbidly religious', prayers and bible readings occurred twice a day, church attendance three times every Sunday, no use of the horses for driving or riding on Sunday and no hot food on Sundays either. His father forbade the family to go to the theatre and all reading material was personally approved.

Francis went to Huntly School, Morton, initially, and his letters home at this time showed his inventive mind was already developing as he drew special devices for use by his mother at home as labour saving gadgets. His secondary

education was at Wanganui Collegiate. At some time around 1912 (when Francis was 20 years old) the family returned to England. The father took up a parish at Billingford in Norfolk and Francis went to St Bartholomew's Hospital to start his medical studies.

### Medical student life

The first 3 years of Francis's medical training were uneventful except for his change of name. The family were originally of Huguenot descent and his father decided to change their name back to the original form namely de Caux. This took place some time during 1914. His fourth year at St Bartholomews took 3 years to complete, since the First World War interrupted his studies; de Caux travelled to France to work as *Medecin Auxiliaire* in the *Service de Sante* at the military hospital at Yvetot, France. He passed his obstetric finals in October 1918, his medicine in July 1920 and his surgery final in 1921 having had to spend another extra year completing his studies.<sup>1</sup> The GMC record him fully registered MRCS LRCP on the 4 February 1921.

### Early medical career

Having completed his house jobs he was appointed resident anaesthetist at St Bartholomews at the end of October 1921, a 6-month post with a salary of £80.00 per annum. In the following month his name appeared in the national press for the first (but not the last) time when a play he had co-authored was banned by the Lord Chancellor. The

---

D. J. Wilkinson, MB, BS, FFARCS, Consultant, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE.

Accepted 6 July 1990.

Parts of this paper were presented at the History of Anaesthesia Society Meeting at Leicester and Southend in 1988 and at the World Congress of Anaesthesiology in Washington, USA in the same year.



*Daily Express*<sup>2</sup> wrote 'Censor bans a lunacy play. Ethics of removing an imbecile child. The censor has placed an absolute ban on a play intended for the London Grand Guignol. It is the first time he has taken such drastic action concerning any playlet submitted for this theatre and the fact that Dr de Caux has collaborated in the authorship makes it even more remarkable. The play, entitled "Euthanasia", deals with the terrible question of whether the parents and a doctor have the right to destroy the life of an imbecile boy who stands in the line of inheritance to a baronetcy'. A subsequent report in December of 1921<sup>3</sup> showed the play had been amended and was acceptable for public performance. This was an extraordinary topic for a young doctor to write about for the London stage and gives some insight into the inquiring mind of de Caux and his willingness, even at this early age, to challenge the moral and social values of his time.

At the end of his residency in June of 1922 he was promoted to senior resident anaesthetist with his salary increased to £150 a year. His diary at this time shows the sort of day he would spend whilst on call: '08.30, awake; 09.15, breakfast; 10.45, GA for tonsillectomy for Hamer Bedford Russell; 12.45, lunch; 13.15, list for Dunhill—five cases; 17.00, list finished; 18.00, gas for Potts; 19.30, thrombosis of lateral sinus for Sydney Scott—died in bed; 22.00, gangrenous appendix for Maingot; 01.00, ectopic gestation for Maingot; 02.30, finished. Bed'.

A long day but not too arduous by today's standards. His duties were varied but he worked with many of the great surgeons of the era. His diary notes performing a medical examination on Lord Horder and on another date how he dined with Dr Bengue, the French anaesthetist, who designed the special canister and valve for the administration of ethyl chloride.

In September of 1922 he was again newsworthy,<sup>4</sup> as a headline read 'Dead man lives again. Astounding feat of surgery—heart massaged. A man whose breathing and heart beats had ceased for over an hour, was restored to life and lived for 27 hours. The amazing feat of modern surgery was performed by Dr H. Bedford Russell, a young Harley Street specialist, on a patient at St Bartholomew's Hospital. The patient was a man of 27 years, suffering from septic tonsils; an operation was decided upon but after one tonsil had been removed the heart and breathing stopped. An astonishing battle for the man's life was then begun by Dr Russell and Dr F. P. de Caux. Pituitrin was injected straight through the chest into the heart. Dr Russell then decided to massage the heart. The abdomen was cut and the heart massaged through the diaphragm for ten minutes. The heart remained motionless, the patient was dead. Quick as a thought, a further injection was made and the heart massaged with hands inside the pericardium, adrenaline was injected and this had a marked effect. Within 50 minutes the heart began to move and at the end of an hour the heartbeat came back and the dead man breathed. "It is the most extraordinary case I have ever known" said Dr de Caux'.

He was reappointed senior resident that December for a further 5-month period. He had a flat as a student in Chancery Lane and maintained this once qualified. This clearly caused some problems as the Governors of Barts added a corollary to their reappointment that de Caux must sleep in the hospital when on call!

In mid 1923 he left St Bartholomew's and became

Assistant Honorary Anaesthetist at the All Saints Hospital, Finchley Road and started to develop his private practice. He moved to 20 Rupert Street off Shaftesbury Avenue and joined the Royal Society of Medicine.

### Early publications

In August 1923, the *Lancet*<sup>5</sup> published de Caux's description of a new oxygen flowmeter and in October of the same year reported a closed drop ether inhaler.<sup>6</sup> This latter device, a modification of one designed by Paluel Flagg, was made by the instrument maker, A. Charles King, then working for Allen and Hanbury's; the friendship that developed between de Caux and King was to have a lasting effect on the specialty of anaesthesia.<sup>7</sup> In June 1924 he wrote the first of many papers on nitrous oxide anaesthesia for dental operations.<sup>8</sup> This was to be the field in which he would excel. His paper described the development of tracheal tubes for use in dental anaesthesia, his use of two tubes in parallel, one to administer the gases and a wider bore tube to permit the expired gases to escape and be scavenged and which was concurrent with the more well known work of Sir Ivan Magill. Dr de Caux was now an Honorary Anaesthetist to the Kensington, Fulham and Chelsea General Hospital, House Anaesthetist to the Royal Dental Hospital and Consulting Anaesthetist to Bexley Cottage Hospital. In October 1924 his name was cited in a divorce case 'Dancing Doctor to Pay' read the headline<sup>9</sup> an event which was soon followed by his marriage to the woman in the case on 4 June 1925. This again suggests his willingness to flout the social conventions of that era. He moved home at this point to 16a New Cavendish Street and his dental practice continued to develop. He became an Honorary Anaesthetist to the Royal Dental Hospital and a Fellow of the West London Medical and Chirurgical Society (Fig. 1). In 1927 he joined the staff of the North Middlesex Hospital, his son Peter was born in October of the same year and he again moved to a larger home at 25 Weymouth Street, W1.

### Influence on A. Charles King

At some point in the late 1920s de Caux visited America and spent time with McKesson who had developed his revolutionary demand flow nitrous oxide-oxygen machine. de Caux was greatly impressed with the apparatus and on returning to England found that his instrument-maker friend Charles King had just gone into business for himself in Devonshire Street. de Caux persuaded him to import the apparatus and records from the McKesson works show that he was the first to use such a machine in England. This started Charles King on his close relationship with anaesthesia that was so beneficial to the specialty. King later modified a McKesson for de Caux and indeed offered this adaptation in one of his catalogues, with full details of use.

### Early use of curare

In 1928 whilst at the North Middlesex Hospital he experimented with an aqueous solution of curare in an attempt to provide increased muscle relaxation during nitrous oxide anaesthesia.<sup>10</sup> His results were inconclusive because he was hampered by a nonstandard product which obviously had a wide variation in clinical effect. He discussed his results



Fig. 1. Dr F.P. de Caux as a young man.

with Dr Victor Goldman who reported them in a subsequent book,<sup>11</sup> but these extraordinary experiments have received little publicity at the time or since. He was the first anaesthetist in this country to attempt to use this drug for this purpose and it is unfortunate that he did not persevere with this line of research and publish his work. He was unable to interest the pharmaceutical industry at that time in this concept despite approaches to several companies.

#### Publications during the early 1930s

The early 1930s for de Caux were characterised by a period of intensive academic effort whilst at the same time he continued to build his private practice, particularly in the dental field. He published a series of papers<sup>12-18</sup> on a variety of topics in all the major journals of that era. Of particular note was his paper on nitrous oxide in oral surgery,<sup>12</sup> which gave details of a personal series of over 20 000 nitrous oxide anaesthetics in a 4-year period, a feat few could equal today. This paper also described his flexometallic tracheal tubes which provoked considerable interest both in this country and in the USA. Ralph Waters wrote to him in 1932 to enquire for further details for a book he was planning to write with Guedel and Rovenstine. de Caux replied in January of 1932 and described his various techniques including the use of two tubes referred to earlier. He states that since using the McKesson machine he had adopted the use of a large-bore tube only, but found it tended to kink as it warmed. He therefore developed a series of wire re-inforced rubber tubes to which he added

the 'air cushions' (cuffs) that Waters had sent him. The tubes were manufactured and sold by Allen and Hanbury's.

#### Coroner's case Scandal

In September of 1932 de Caux was asked by a colleague to give a second opinion on a patient who had undergone surgery for an abortion. The patient had bled postoperatively and developed septicaemia. de Caux arranged for her urgent admission into a London nursing home and set up her medical care to be provided by two other practitioners. The patient died and the press became heavily involved when her mother and a Russian woman doctor friend of the family committed suicide. The coroner's summing up was fully reported and was particularly hostile to Dr de Caux. The *Daily Express*<sup>19</sup> wrote 'Mr Roddy (the Coroner) said "Obviously it is a dangerous case, and probably a criminal one, and so long as the law regards criminal abortion as a most serious felony in regard to the person who procures it, and in procuring it causes death as being guilty of murder, it is the duty of the coroner to endeavour to find out who that person was". He continued "—the person in charge of the case, Dr de Caux, takes no part in seeing that patient and leaves her in the hands of three other doctors, and does not sign the death certificate. I have indicated in my questions to him what my view is as to his conduct in this case which bristles with suspicion. I have a definite suspicion in my mind as to what exactly took place but it cannot be proved by direct evidence and the only course I can pursue is to record a verdict of death from septic abortion and leave the verdict open"'.<sup>20</sup>

At this distance in time it is impossible to assess de Caux's actual involvement in the case but the event certainly marked him both socially and professionally. From this period onwards he started to be treated warily by his colleagues and his academic publications diminished. He was still able to expand his private practice, however, and was appointed Honorary Anaesthetist to the Gordon and Prince Beatrice Hospital as well as to the Dreadnought Hospital, Greenwich in 1934.

#### Trip to Russia

In August of 1934 he travelled to Russia as part of a medical mission. He travelled by boat, the *Sibir*, to Leningrad and visited a variety of tourist sites as well as hospitals and educational and research institutes. Notable amongst the latter was the Pavlov Institute. He then travelled by train to Moscow and visited further medical and nonmedical sites. He gave one anaesthetic at the Bodkin Hospital for a Professor Solovov, a nephro-ureterectomy for tumour which took 1.5 hours. de Caux noted that the patient had received no pre-operative preparation 'and so a little ether had to be added to the gas and oxygen, but not much'. He then returned home via Berlin and went back to work.

#### de Caux and Abortions

This trip to Russia had a profound effect on de Caux. During his stay he had seen the Russian approach to abortion which was freely available on demand, indeed at that time it was used as a form of birth control as other methods were not available. He wrote a paper on the

subject which was published in 1935<sup>20</sup> which highlighted the discrepancies between the English and Russian approaches. It was at this time that de Caux certainly started to provide an abortion service in London; whether he had performed the odd case or two before is unknown but he was now convinced of both the rightness of such procedures and the wrongness of the law trying to prevent them.

He moved home to Green Street in Mayfair and also bought a large country home near Marden in Kent. Abortions were performed in both these residences and in fact his country home was partly converted into a nursing home. This practice soon became widely known in medical circles in London and de Caux became even more distanced from his professional colleagues.

His general anaesthetic practice still continued, and de Caux continued to lecture and attend meetings. In 1936 he travelled to Vienna and spoke at the 9th International Dental Congress on nitrous oxide anaesthesia. He corresponded regularly with many of the leaders of the anaesthetic world at that time including Macintosh, Mushin and Goldman, who often sought his advice over matters relating to nitrous oxide anaesthesia. This was the time when there was a strong swell of public opinion towards a change in the law to permit abortions in cases of medical need. The Birkelt Report of 1936 and a report produced by an interdepartment committee in 1937 attempted to analyse a considerable amount of evidence relating to abortion, most of which was at variance. Many doctors at this time pleaded for clarification of the law but were ignored. No use was made of the findings of these two committees and then in 1938 came the Aleck Bourne case. Mr Bourne was an eminent gynaecologist of that time, and when confronted with a 14-year-old girl who had been raped by several soldiers, performed a termination. He informed the authorities of the facts of the case and was prosecuted and then acquitted (*Rex v. H. Bourne*). Public opinion was largely with Mr Bourne because his defence rested on the mental health implications to the mother. Despite this legal precedent no change in the actual law had taken place, each individual case had to be considered on individual merit. There was an element of uncertainty remaining and each doctor and policeman was forced to bear considerable responsibility in interpretation of the law.<sup>21</sup>

The concepts of therapeutic abortion to preserve the mental or physical well being of the mother received general public approval in extreme cases such as the Bourne case, but the idea of abortion on demand for personal convenience was not generally accepted. Despite this ambiguity in public opinion the law was still clear and de Caux, a highly intelligent man, must have been under no illusion as to what risks he was running. Prosecutions were taking place on a regular basis and de Caux was under considerable threat.

#### de Caux and the law

de Caux's abortion practice fell under police surveillance soon after the start of the Second World War. His house in Kent was in a residential area of the south coast and all vehicles passing into the zone were stopped and the names and addresses of all on board noted. de Caux's regular trips on a Friday evening with a variety of woman companions, who then returned at differing times after the weekend were

soon noticed. The police traced a number of women who had visited de Caux's nursing home and eventually several confessed to having undergone terminations.

In July of 1942 de Caux was remanded at Cranbrook Assizes in Kent for performing abortions and was subjected to a very public trial at the Old Bailey in September of the same year. He was found guilty on eight counts and sentenced to 5 years' jail. The popular press at this time made much of the scandal and his name was widely broadcast. His jail sentence was passed at Camp Hill prison on the Isle of Wight. de Caux worked in the prison library which must have provided some solace. In February of 1943 his name was erased from the medical register by the GMC and his LRCP MRCS qualifications rescinded.

#### Aftermath of prison

de Caux left prison on 18 January 1946 and found he had re-entered the world during the post war depression. He had no means of deriving a living since he was no longer regarded as qualified and was not on the medical register. His home had been sold whilst in prison and the solicitor appointed by the Crown to oversee his affairs had not acted correctly and de Caux was forced to sue him. His son had moved to America in 1939, had entered Yale and was doing well. His wife decided to divorce him soon after he left prison in December 1947.

Employment at this time was not easy to find. de Caux approached many of the anaesthetic and drug manufacturing companies but no posts were vacant for him. He made a series of applications to the Royal Colleges of Surgeons and Physicians for re-instatement, but despite letters of support from a variety of eminent doctors, these applications were refused. de Caux was naturally upset and greatly disappointed by these failures since he believed he still had a role to play in medicine and particularly in anaesthesia. Many of his dental friends asked him to give occasional anaesthetics for them and this he did despite his lack of official qualifications. These actions undoubtedly upset the medical boards at that time and must have severely prejudiced his chances of reinstatement.

#### Writing career

In the period before going to prison, whilst in jail and in this post war era he wrote a textbook of anaesthesia predominantly relating to nitrous oxide anaesthesia. This book gives a detailed historical review based on personal interviews made by de Caux with many who could recall the earliest days of the specialty. The book continues to expand his theories and techniques of practice for nitrous oxide anaesthesia and was a highly erudite study of that time. Part of the book was reviewed by Professor Mushin in Cardiff but never came to publication as de Caux turned away from his former specialty.

In an attempt to provide a living he became a medical journalist and during 1947 travelled as the British Medical Representative of the *Chinese Medical Journal* and the *National Medical Journal of China*.

#### Future career

de Caux then became interested in alternative medical practice. He went to Amsterdam in 1948 and attended



Fig. 2. Dr F.P. de Caux in later life.

Samuels' clinic there and learnt of his cancer therapy techniques. This involved a method of electrostimulation and hormone injections. de Caux learnt all he could and then set up a practice in Devonshire Street. This practice was slow in developing and de Caux made occasional enquiries about restarting again as a medical student, or emigrating back to New Zealand, South Africa or America. None of these projects was successful; neither was an attempt to become a pharmacist. His cancer treatments had some success, however, and the practice slowly developed. In 1951 he wrote a book on the subject, *A New Light on Cancer*, which reviewed his methods and results.<sup>22</sup>

#### Later life

de Caux purchased property in Brighton and spent his weekends there, travelling up to town for his practice during the week. In his sixties he met the woman who would become his second wife in 1960. As he grew older his health started to fail. He had problems with a slipped disc, developed hypertension and ischaemic heart disease and was overweight when he underwent a prostatectomy at the Lambeth Hospital in 1965. He developed thrombophlebitis postoperatively and suffered a fatal pulmonary embolus.

His young wife had his body cremated at the Downs Crematorium in Brighton and his ashes were scattered on the Garden of Remembrance there. His passing was unnoticed by the medical press or members of his original profession.

#### de Caux in perspective

de Caux was a great anaesthetist of his era, well respected by his peers and by those he taught who went on to

influence their chosen specialty; of this latter group, Victor Goldman and William Mushin still regard him highly. He was a highly intelligent doctor, widely read, an innovator who produced useful apparatus and who also performed some unique experiments with muscle relaxants.

Why he chose to step outside the law and perform terminations illegally is difficult to understand, difficult that is until his life story is considered. His 'morbidly religious' and restrictive childhood prompted a very open outlook in later life. As a young man he was not afraid to challenge social customs which he considered outmoded. His trip to Russia undoubtedly influenced him deeply and encouraged him to take up the challenge of those women who needed terminations for social reasons. He acted, I believe, out of altruism rather than for self gain. It is tragic to consider the punishment he received for acting in a manner which by today's reckoning would be unremarkable. He did not live to see the law changed by the Abortion Act but how amused he would be to see his own teaching hospital performing lists of terminations every week.

Francis Percival de Caux's contributions to medicine deserve a wider recognition. Sir Ivan Magill described him as 'the finest exponent of gas and oxygen anaesthesia that I ever saw,' no mean accolade. He was pioneer in anaesthesia, in research into the specialty and also in challenging social dogma he believed inappropriate. Such a considerable contribution should not pass unrecognised.

#### Acknowledgments

I would like to thank the following people and institutions for their considerable help in the research and preparation of this paper: Mrs R. de Caux, Dr V. Goldman, G. Yeo, the Daily Express Newspaper Archive, the British Museum Newspaper Archive and also Miss M. Langley for her considerable secretarial help. I am grateful to the Department of Medical Illustration at St Bartholomew's Hospital for the illustrations shown.

#### References

1. St Bartholomew's Hospital archive records. (a) Student Signature Books M.S.1./3; (b) Registers of Attendance of Students. M.S. 54/1.
2. *Daily Express*. 7 November 1921.
3. *Daily Express*. 3 December 1921.
4. *Sunday Express*. 10 September 1922.
5. DE CAUX FP. A new pattern oxygen sight feed valve. *Lancet* 1923; **2**: 228.
6. DE CAUX FP. Closed drop ether inhaler. *Lancet* 1923; **2**: 789-90.
7. WILKINSON DJ. A. Charles King: a unique contribution to anaesthesia. *Journal of the Royal Society of Medicine* 1987; **80**: 510-4.
8. DE CAUX FP. Endotracheal gas and oxygen anaesthesia in dental surgery. *The Ivory Cross Journal* 1924; **6**: 643-5.
9. *Daily Express*. 30 October 1924.
10. WILKINSON DJ. Dr FP de Caux—the first user of curare for anaesthesia in England 1990. *Anaesthesia* 1991; **46**: 49-51.
11. GOLDMAN V. *Aids to anaesthesia*, 2nd edn. London: Baillière Tindall and Cox, 1948: 118-9.
12. DE CAUX FP. Recent advances in methods of administering nitrous oxide in oral surgery. *British Dental Journal* 1930; **51**: 471-84.
13. DE CAUX FP. Glucose—when and how to use it. *Anesthesia and Analgesia* 1930; **9**: 203-7.
14. DE CAUX FP. Recent advances in methods of administering nitrous oxide in oral surgery. *Anesthesia and Analgesia* 1930; **9**: 254-64.

15. DE CAUX FP. System of anaesthesia for the dental anaesthetist. *British Journal of Anaesthesia* 1931; **9**: 22–40.
16. DE CAUX FP. Premedication. *Anesthesia and Analgesia* 1932; **11**: 68–70.
17. ABEL AL, DE CAUX FP. Nitrous oxide and oxygen anaesthesia. *Clinical Journal* 1932; **61**: 436–41.
18. DE CAUX FP. Anesthesia for the dental surgeon. *Anesthesia and Analgesia* 1933; **12**: 204–7.
19. *Daily Express*. 29 September 1932.
20. DE CAUX FP. Birth control in Soviet Union and in Great Britain. *Marriage Hygiene* 1935; **2**: 170–9.
21. CHESSE E. The Doctor's Dilemma. *New Statesman*, 7 June 1958.
22. DE CAUX FP. *A new light on cancer*. London: Carroll and Nicholson, 1951.

## Forum

### Intubation guide marks for correct tube placement

#### A clinical study

S. Mehta, FFARCS, Consultant, Department of Anaesthetics, Burnley General Hospital, Burnley, Lancs BB10 2PQ.

#### Summary

*The tracheas of 140 adult patients were intubated with either TFX or Portex tracheal tubes. Guide marks were printed at variable distances proximal to the tube cuffs, and during intubation the guide mark was positioned at the level of the vocal cords. The distance between the bevel end of the tube and the carina was determined with a fiberoptic bronchoscope. The mean distance between the tip of the tracheal tube and the carina varied between 3.7 and 4.1 cm with the head in the neutral position. The tip of the tracheal tube approaches the carina by a mean distance of 0.5 cm when the head is moved from the extended position to the neutral position. It is recommended that a guide mark be placed 2.5 cm from the proximal end of the cuff in tubes used for adult males and 2.25 cm in tubes used for adult females. The use of guide marks is a simple, safe and reliable method for correct tracheal tube placement.*

#### Key words

*Intubation, tracheal; technique.*

Inadvertent bronchial intubation due to accidental migration of the tube tip into a main stem bronchus is a major complication of tracheal intubation. This is a particular hazard for mobile patients in the ICU. In adult patients, the tracheal tube moves an average of 1.9 cm towards the carina with flexion of the neck from a neutral position and a similar distance away from the carina with extension.<sup>1</sup> Thus the mean tube movement between flexion and extension is about one third to one fourth the length of the normal adult trachea. This movement occurs regardless of the route of intubation or inflation status of the cuff. To minimise the possible adverse effects of tube malposition, Conrardy *et al.*<sup>1</sup> have recommended that the tip of the tracheal tube be placed in the middle third of the trachea with the neck in a neutral position.

Malposition of the tracheal tube can also cause vocal cord paralysis and accidental extubation. Laryngeal paralysis may follow as a result of compression of the anterior branch of the recurrent laryngeal nerve between the cuff and the thyroid lamina when the cuff is inflated within the larynx. The suspected point of vulnerability from cuff damage is located between 6 and 10 mm below the posterior end of the free edge of the vocal cord. This mechanism of true vocal cord paralysis following tracheal intubation was first described by Cavo.<sup>2</sup> He emphasised the importance of proper tube placement and advocated the use of a mark on the tracheal tube 1.5 cm above the upper end of the cuff to prevent vocal cord paralysis.

A recent study on 50 cadavers revealed that the mean distance between the vocal cords and lower border of the cricoid cartilage in men was 3 cm and in women 2.75 cm.<sup>3</sup> It was suggested that if manufacturers of tracheal tubes put a circular mark on the tube 3 cm proximal to the cuff,

anaesthetists could position the cuff just below the cricoid cartilage.<sup>4</sup> This would not only prevent accidental bronchial intubation but also true vocal cord paralysis. The present study was designed to examine whether appropriately placed guide marks proximal to the cuff can help position the tip of the tracheal tube in the middle third of the trachea and thus prevent these complications.

#### Methods

One hundred and forty adult patients who were to undergo general surgical procedures participated in the study. The inclusion criteria were freedom from disabling illness (ASA grade 1 or 2), age between 24 and 80 years and willingness to give written consent. Those patients in whom vocal cords could not be visualised were not studied. Following induction of anaesthesia and muscle relaxation the tracheas of all patients were intubated with either a TFX tracheal tube with a Sensitive large volume, low pressure cuff or Portex tracheal tube with a Profile cuff. Male patients were intubated with 8.00 mm internal diameter tubes, while 7.00 mm internal diameter tubes were used in female patients.

The manufacturer of TFX tracheal tubes printed a 2-mm wide circular guide mark 30 mm proximal to the cuff on 8.00-mm tubes and 27.5 mm on 7.00-mm internal diameter tubes, while on all Portex tubes a guide mark of similar width but only a quarter of the circumference around the tube was printed 30 mm proximal to the cuff (Fig. 1).

During intubation the guide mark on the tube was positioned at the level of the vocal cords. The tracheal tube was then secured in place with a cotton bandage. With the head in the neutral position the fiberoptic bronchoscope

S. Mehta, FFARCS, Consultant Department of Anaesthetics, Burnley General Hospital, Burnley, Lancs BB10 2PQ.

Accepted 10 July 1990.



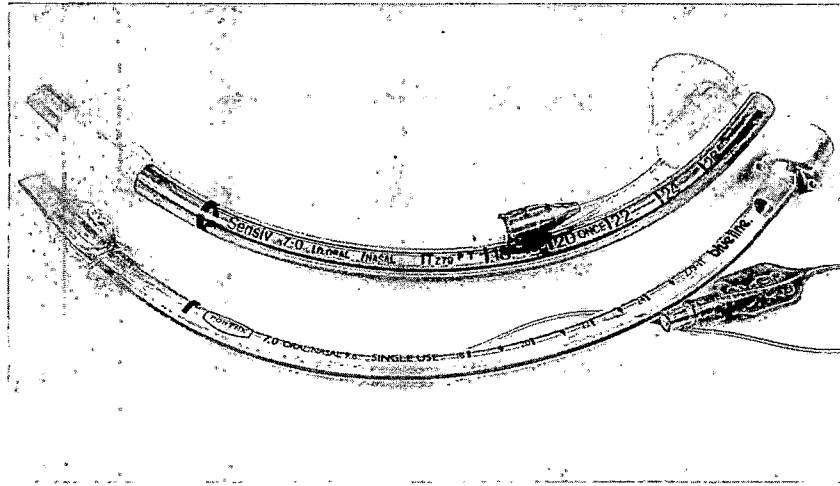


Fig. 1. Guide marks on TFX and Portex tracheal tubes.

was introduced into the tracheal tube and the distance between the bevel end of the tracheal tube and the carina measured (Fig. 2). Finally, in 50 patients the distance between the tip of a TFX tracheal tube and the carina was also measured with the head extended at the atlanto-occipital joint.

### Results

The details of the patients studied are given in Table 1. The patients in both the TFX and Portex groups are matched as regards sex, age, weight and height. Table 2 shows the mean distance between the proximal end of the cuff and the tube tip in both the TFX and Portex tubes. Table 3 shows the mean distance between the tip of the tracheal tube and the carina with the head in the neutral position, this varied between 3.7 and 4.1 cm and was significantly shorter with the Portex tubes in female patients.

Table 4 shows the mean distance between the tip of the TFX tube and the carina when the head is in the extended position. The tip of the tracheal tube moves towards the carina by a mean distance of 0.5 cm when the head is moved from the extended position to the neutral position.

### Discussion

Tracheal tubes are in everyday use, not only by anaesthetists but by doctors working in accident units, high dependency and intensive care and are, in many cases, essential to the safety of patients. It is, therefore, appropriate to

consider additional safety features which could make them safer to use. It would be an advantage if one could, with confidence, place the tip of the tracheal tube precisely in the middle third of the trachea as suggested by Conrady.<sup>1</sup>

In the past several workers have described various methods to prevent inadvertent bronchial intubation. The Cole pattern paediatric tube incorporates a shoulder to prevent deep placement.<sup>5</sup> One of the problems with the Cole design is laryngeal damage with prolonged intubation. Morgan and Steward calculated the length of an orotracheal tube from the equation based on the height of the patient.<sup>6</sup> Other methods indicating or limiting the level of descent of the tracheal tube include the use of magnetic markers,<sup>7</sup> laryngeal hooks,<sup>8</sup> cuff palpation,<sup>9</sup> transtracheal illumination,<sup>10</sup> positioning the tube by referencing the mark on the side of the tube<sup>11</sup> and X rays. Some of these methods are either unreliable or require the use of additional equipment.

There are three factors that determine the position of the tube in the trachea. These are, firstly, how far the tube is advanced beyond the vocal cords. Secondly, the position of the head and neck in relation to the trunk and finally the length of the airway between the vocal cords and the carina. The length of the tube advanced beyond the vocal cords is determined by the position of the intubation guide mark on the tube, length of the cuff and distance between

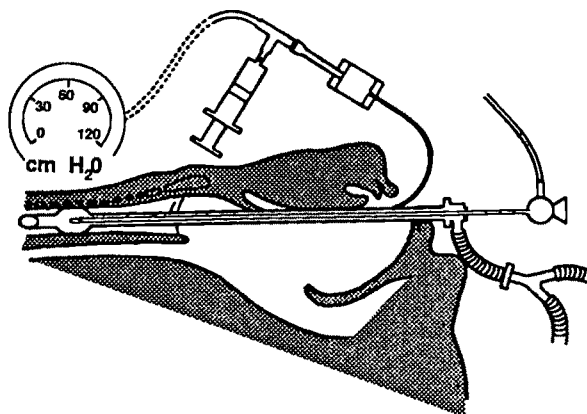


Fig. 2. Schematic representation of technique.

Table 1. Demographic details (SD).

	TFX	Portex
Total	70	70
Male: female	35:35	35:35
Mean age; years	41.6 (12.2)	40.4 (11.6)
Range	24-80	26-81
Mean weight; kg	62.4 (13.4)	65.1 (10.5)
Mean height; cm	158.2 (12.2)	157.4 (13.0)

Table 2. Distance between proximal end of the cuff and the tube tip. Values expressed as mean (SD).

Size of Tube (ID)	TFX	Portex
	Distance in mm	
7 mm	58 (2)	60 (2)
8 mm	63 (2)	65 (2)

**Table 3.** Distance between the tip of the tracheal tube and the carina with head in neutral position. Values expressed as mean (range).

Sex	Size of tube (internal diameter)	TFX	Portex
		Distance in cm	
Female	7.0 mm	4.0 (2.5–5)	3.7 (2.2–4.8)
Male	8.0 mm	4.1 (2.8–5.3)	4.0 (2.4–5.1)

**Table 4.** Distance between the tip of TFX tube and the carina with head extended. Values expressed as mean (range).

Sex	n	Distance in cm
Female	25	4.5 (3–5.5)
Male	25	4.6 (3.2–5.6)

the cuff and the tip of the tracheal tube. Reduction in both the length of the cuff and distance between the cuff and the tip of the tube will further help to place the tube in the correct position and prevent complications due to malposition.

This study confirms that intubation guide marks provide a reliable method of placing the tip of the tracheal tube in the correct position in the trachea. These guide marks will allow the tip of the tracheal tube to remain 3.7 to 4.1 cm above the carina. However, in three female patients who were intubated with the Portex tube, the tip of the tracheal tube was found to be only 2.2 cm from the carina. These patients may be considered at high risk for bronchial intubation. It was possibly due to the fact that guide marks on 7.0 mm internal diameter Portex tubes were placed 3 cm from the proximal end of the cuff. It is evident from the study that the tube tip advanced an average of 0.5 cm towards the carina when the head is moved into the neutral position following intubation.

We therefore recommend that guide marks should be placed 2.5 cm from the proximal end of the cuff in tubes used for adult males and 2.25 cm for adult females. This

will still allow the cuff to lie in the trachea while the head is in the neutral position and prevent vocal cord paralysis due to compression of the anterior branch of recurrent laryngeal nerve between the cuff and the thyroid lamina.

The limitation of this technique is that it can only be used when the vocal cords are visible. It is hoped that use of intubation guide marks on the tracheal tubes will markedly reduce the frequency of inadvertent bronchial intubation and prevent vocal cord paralysis and accidental extubation.

#### Acknowledgments

I thank Portex and TFX Ltd for their assistance with the development of this project. I also acknowledge the assistance and cooperation of members of the Anaesthetic Department and nursing staff, and thank Mrs J. Watson for typing the manuscript.

#### References

1. CONRARDY PA, GOODMAN LR, LAINGE F, SINGER MM. Alteration of endotracheal tube position. Flexion and extension of the neck. *Critical Care Medicine* 1976; **4**: 7–12.
2. CAVO JW JR. True vocal cord paralysis following intubation. *Laryngoscope* 1985; **95**: 1352–9.
3. MEHTA S. Endotracheal intubation: friend or foe? *British Medical Journal* 1986; **292**: 694.
4. MEHTA S. Safer endotracheal intubation. *Lancet* 1986; **1**: 1148.
5. COLE F. A new endotracheal tube for infants. *Anesthesiology* 1945; **6**: 87–8.
6. MORGAN GAR, STEWARD DJ. A preformed paediatric orotracheal tube design based on anatomical measurements. *Canadian Anaesthetists' Society Journal* 1982; **29**: 9–11.
7. MCCORMICK WF, CRONE RK. Pediatric tracheal intubation system that may reduce X ray use: Preclinical assessment. Proceedings of the eighth annual conference of the IEEE/Engineering in Medicine and Biology Society, November 1986; 140–3.
8. MACNEIL A. An orotracheal tube with laryngeal hooks. *Anaesthesia* 1988; **43**: 803.
9. EHRENWERTH J, NAGLE S, HIRSH N, LA MANTIA K. Is cuff palpation a useful tool for determining endotracheal tube position? *Anesthesiology* 1986; **65**: A137.
10. MEHTA S. Transtracheal illumination for optimal tracheal tube placement. *Anaesthesia* 1989; **44**: 970–2.
11. OWEN RL, CHENEY FW. Endobronchial intubation: A preventable complication. *Anesthesiology* 1987; **67**: 255–7.

### A comparison of lignocaine with prilocaine in axillary brachial plexus anaesthesia

E.P. McCoy,\* MB, BCh, BAO, Senior House Officer, C.M. Wilson, MD, FFARCSI, Consultant, Anaesthetic Department, Ulster Hospital, Dundonald, Belfast BT16 0RH, Northern Ireland

#### Summary

Twenty patients received either lignocaine 1.5% with 1/200 000 adrenaline (group L), or prilocaine 1.5% plain (group P) as a brachial plexus block for surgery to the upper limb, in a randomised double-blind study. The two groups were comparable in age, weight and duration of surgery and there were no significant differences between the two groups with regard to onset, pattern or degree of sensory loss. The degree of motor loss was also comparable. The group L patients had a statistically significant longer duration of sensory loss than those in group P. All the blocks were performed using the same technique and provided complete surgical anaesthesia. Prilocaine 1.5% plain provides adequate sensory and motor blockade for brachial plexus anaesthesia and is a suitable agent for medium duration surgery to the upper limb.

#### Key words

Anaesthetic techniques, regional; brachial plexus.  
Anaesthetics, local; lignocaine, prilocaine.

Various methods of brachial plexus block are employed; the two most popular are the parascapular and axillary techniques.<sup>1,2</sup> Axillary brachial plexus block is a safe, relatively simple method of anaesthetising the upper limb.<sup>2</sup> Lignocaine 1.5%, mixed with adrenaline is an agent commonly used and provides surgical anaesthesia for between 2 and 4 hours. Adrenaline is added to lignocaine to reduce the plasma levels, minimise the risk of systemic toxicity and prolong the duration of block.<sup>3</sup>

The local anaesthetic agent prilocaine has a high metabolic clearance, which results in low plasma levels and consequently low systemic toxicity; it therefore possesses a safety margin greater than that of the more commonly used local anaesthetics.<sup>4</sup> It has been considered the drug of choice for medium duration procedures when a large dose of local anaesthetic is required.<sup>5</sup> It does not require the addition of adrenaline so it may be the agent of choice in this procedure. However, for brachial plexus blockade, the commercially available 1% solution does not provide adequate muscle relaxation.<sup>6</sup>

The purpose of this study was to compare a 1.5% preparation of prilocaine plain with lignocaine 1.5% and adrenaline (1/200 000) in brachial plexus anaesthesia, for clinical efficacy and the extent and duration of sensory and motor blockade.

#### Method

The study had the approval of the hospital medical ethics committee. Written informed consent was obtained from 20 ASA grade 1 and 2 unpremedicated patients listed for surgery to the hand. They were allocated at random to one of two groups. Group L received axillary brachial plexus anaesthesia with 30 ml of 1.5% lignocaine with adrenaline, group P received 30 ml of prilocaine 1.5% plain. The study was double-blind in that all the blocks were carried out by the investigator (E.McC.), who was unaware which agent was used. The assessor (C.W.) was also unaware which agent was used.

The apparatus consisted of a 22 SW gauge (Becton and Dickinson) regional block needle connected to an extension set (Vygon), which was primed with the study drug. Two syringes containing the full dose of local anaesthetic were available.

Lignocaine 1.5% with 1/200 000 adrenaline was freshly prepared by mixing 20 ml of lignocaine 1% with 1/200 000 adrenaline (Astra) and 20 ml of lignocaine 2% with 1/200 000 adrenaline (Astra). Thirty ml of this mixture was used. Prilocaine 1.5% plain was prepared in the same volume (30 ml), by diluting 11.25 ml of commercially available prilocaine 4% (Citanest 4%) in 18.75 ml of normal saline.

Patients were prepared in the normal way. In the operating theatre the patient was placed in a supine position and an electrocardiogram, pulse oximeter and noninvasive blood pressure cuff were applied. When venous access had been secured in the contralateral arm, axillary brachial plexus block was carried out. No attempt was made to specifically block the musculocutaneous nerve. After completion of the block a cuff was inflated on the upper arm to 200 mmHg for 10 minutes.

A single-shot technique was employed because it was thought surgery would last no more than 3 hours. In the event of prolonged surgery or inadequate blockade at any time it was planned to convert to general anaesthesia, and the patient was prepared for this. No patient required general anaesthesia.

Sensory changes were assessed with a 25 SW gauge needle 15, 25 and 40 minutes after insertion of the test drug in the areas supplied by the following nerves: axillary, musculocutaneous, radial, median, ulnar, medial cutaneous nerve of arm and medial cutaneous nerve of forearm. The results were recorded using a scale of 0–2; 0 = no sensory loss; 1 = loss of pinprick sensation; 2 = complete loss of sensation.

The power of the arm was assessed 40 minutes after injection of the test drug. The results were recorded on a scale of 0–3; 0 = no motor loss; 1 = slight loss of power;

\*Current address: Anaesthetic Department, Altnagelvin, Londonderry BT47 1SB, Northern Ireland  
Accepted 11 June 1990.

**Table 1.** Patient characteristics, mean (SEM).

	Group L (n = 10)	Group P (n = 10)
Age; years	42.4 (5.5)	43.5 (4.7)
Weight; kg	68.0 (2.4)	68.2 (3.3)

**Table 2.** Type of surgery.

	Group L	Group P
Carpel tunnel	4	2
Tendon repair	5	4
Nerve repair	0	1
Laceration	1	3

**Table 3.** Duration of surgery in each group.

	Group L	Group P
< 1 hour	2	1
1-2 hours	6	7
2-4 hours	1	1
4-6 hours	1	1

2 = very weak power; 3 = complete loss of power. No peri-operative sedation was employed.

Also recorded were the site, type and duration of surgery and the incidence of complications. The duration of anaesthesia was recorded (in minutes) from the time of insertion of the block to the time at which the patient first experienced sensation at the wound site.

**Table 5.** Degree of motor block in each group at 40 minutes.

Grade (0-3)	Group L	Group P
0	0	0
1	0	0
2	7	6
3	3	4

Twenty-four hours after surgery, the patient's own opinion of the quality of the block was recorded as, excellent, good, fair or poor.

Statistical analysis of sensory loss was performed using Fisher's Exact Probability test. The Mann-Whitney *U* test for nonparametric data was used to compare duration of blockade.

### Results

The two groups were comparable with regard to age, weight, type and duration of surgery (Tables 1, 2 and 3). The time to onset of sensory blockade was also similar in both groups; maximum blockade was at the 40-minute assessment time (Table 4). There were no significant differences between the two groups at any of the sites tested at 15, 25 or 40 minutes. The pattern of blockade in both groups was comparable; loss of sensation was least in the area supplied by the axillary nerves (Table 4). Surgical analgesia was obtained in all cases, with no requirement for individual nerve block, local infiltration or general anaesthesia. Motor blockade in both groups was always either grade 2 (weak) or grade 3 (complete motor block) (Table 5). The degree of motor block did not correlate with the degree or duration of sensory loss. The quality of the blockade was rated as excellent by 19 of the 20 patients and good by one patient.

**Table 4.** Degree of sensory loss.

Nerve area	Grade (0-2)	Time after insertion of block (minutes)					
		15		25		40	
		L	P	L	P	L	P
	n =	10	10	10	10	10	10
Axillary	0	7	7	7	5	6	4
	1	1	1	1	3	2	3
	2	2	2	2	2	2	3
Musculocutaneous	0	1	1	0	0	0	0
	1	3	3	2	0	1	0
	2	6	6	8	10	9	10
Radial	0	1	1	0	0	0	0
	1	3	2	1	2	1	2
	2	6	7	9	8	9	8
Median	0	1	2	0	0	0	0
	1	3	2	2	2	2	2
	2	7	6	8	8	8	8
Ulnar	0	0	1	0	1	0	0
	1	1	3	0	0	0	0
	2	9	6	10	9	10	10
Medial cutaneous nerve of arm	0	0	0	0	0	0	0
	1	2	0	1	0	0	0
	2	8	10	9	10	10	10
Medial cutaneous nerve of arm	0	0	2	0	2	0	2
	1	2	1	1	1	0	0
	2	8	7	9	7	10	8

Table 6. Time to first sensation (hours).

Patient	Group	
	L	P
1	5.75	2.5
2	3.0	1.5
3	3.0	2.0
4	1.75	1.5
5	4.5	1.75
6	6.5	2.25
7	3.5	2.0
8	3.25	2.25
9	4.25	2.5
10	3.25	2.0
Mean	3.9	2.3

The only significant finding in the study was a longer duration of sensory blockade in the group of patients who received lignocaine compared with those who received prilocaine (Table 6) ( $p < 0.01$ ).

### Discussion

The axillary technique of brachial plexus blockade is both safe and reliable,<sup>2</sup> but with this approach, haematoma formation, vascular spasm and inadvertent intravascular injection have all been reported.<sup>7</sup> Unlike other approaches, however, there is no risk of pneumothorax.<sup>8</sup> Previous studies have recorded a success rate of over 90%.<sup>9</sup> In this study there were no failures and no complications.

The risk of administration of toxic total doses of local anaesthetic and accidental intravascular injection remains. Previous studies on brachial plexus anaesthesia have concentrated on technique, and the agent employed was largely ignored. As large volumes are administered, the serum concentrations seen after injection of local anaesthetic may be close to the convulsive threshold with some agents.<sup>10</sup>

The most widely used agent for brachial plexus anaesthesia in the United Kingdom is lignocaine, usually with the vasoconstrictor adrenaline, in concentrations of between 1/80 000 and 1/200 000. With adrenaline, the maximum dose for local infiltration is 7 mg/kg, (3 mg/kg without adrenaline).<sup>11</sup> Although the risk of toxicity is not great, cardiovascular, respiratory and central nervous complications may occur. Accidental intravascular administration may result in cardiac arrhythmias.<sup>11</sup>

Prilocaine is similar in structure and actions to lignocaine, but is less likely to produce toxic effects. It gives consistently lower plasma levels (approximately 35%) than lignocaine;<sup>3</sup> the reason for this would appear to be differences in distribution and metabolism. It is metabolised by amidase in the liver, kidney and lungs. The rapid production of oxidation products, particularly o-toluidine and nitrosotoluidine, may give rise to methaemoglobinaemia.<sup>12</sup> This is usually only important in the presence of severe anaemia or circulatory failure.<sup>4,13</sup> Clinically significant methaemoglobinaemia occurs only following excessive doses, and is readily reversible with methylene blue.<sup>13</sup>

Larger maximal doses of prilocaine can be given without adrenaline than lignocaine with adrenaline.<sup>14</sup> Excessive doses may lead to cardiovascular and central nervous toxicity.<sup>11</sup> Prilocaine is most useful when a high concentration, or a large volume of local anaesthetic is needed, and

the need to add a vasoconstrictor is avoided.<sup>15</sup> The maximum dose of prilocaine is approximately 10 mg/kg.<sup>11</sup>

In this study there was loss of some sensation in all the areas tested. The pattern of sensory loss correlated with previous studies; the musculocutaneous and axillary nerves were the most resistant to anaesthesia in both groups (Table 4).<sup>7</sup> The degree of sensory loss was comparable in both groups. In this study 13 of the 20 operations lasted between 1 and 2 hours and 15 lasted less than 4 hours. The mean duration of sensory blockade in the lignocaine group (3.9 hours) was significantly longer than the duration of sensory block in the prilocaine group (2.3 hours) ( $p < 0.01$ ). This duration of anaesthesia is more than adequate for most surgical procedures to the upper limb. This study also demonstrated that prilocaine 1.5% plain was as effective as lignocaine 1.5% with adrenaline in achieving motor blockade (Table 5).

Prilocaine 1.5% plain would appear to be the agent of choice in brachial plexus blockade for surgery lasting less than 2 hours. Unfortunately, a preparation in this concentration is not yet commercially available.

### Acknowledgements

The authors thank Mr S. Barnes and Dr P. White for their help in conducting this study, and Drs P. Loughran and P. Hawthorne for advice.

### References

- VONGVISES P, PANJAYANOND T. A parascapular technique of brachial plexus anaesthesia. *Anesthesia and analgesia ... Current Researches* 1979; **58**: 267-73.
- DE JONG RH. Axillary block of the brachial plexus. *Anesthesiology* 1961; **22**: 215-25.
- SCOTT DB, JEBSON PJR, BRAID DP, ÖRTENGREN B, FRISCH P. Factors affecting plasma levels of lignocaine and prilocaine. *British Journal of Anaesthesia* 1972; **44**: 1040-9.
- LUND PC, QUICK JC. Propitocaine (Citanest) and methaemoglobinemia. *Anesthesiology* 1965; **26**: 569-71.
- MACLEAN D, CHAMBERS WA, TUCKER GT, WILDSMITH JAW. Plasma prilocaine concentrations after three techniques of brachial plexus blockade. *British Journal of Anaesthesia* 1988; **60**: 136-9.
- HUGHES TJ, DESGRAND DA. *Principles and practice of regional anaesthesia*. New York: Churchill Livingstone, 1987.
- LANZ E, THEISS D, JANKOVIC D. The extent of blockade following various techniques of brachial plexus block. *Anesthesia and Analgesia* 1983; **62**: 55-8.
- YOUSSEF MS, DESGRAND DA. Comparison of two methods of axillary brachial plexus anaesthesia. *British Journal of Anaesthesia* 1988; **60**: 841-4.
- BRAND L, PAPPER EM. A comparison of supraclavicular and axillary techniques for brachial plexus blocks. *Anesthesiology* 1961; **22**: 226-9.
- WILDSMITH JAW. Intravenous regional anaesthesia or axillary brachial plexus block? *Anaesthesia* 1985; **40**: 83.
- GILLES HC, ROGERS HJ, SPECTOR RG, TROUNCE JR. *A textbook of clinical pharmacology*. London: Edward Arnold, 1986.
- SMITH G, AITKENHEAD AR. *Textbook of anaesthesia*. Edinburgh: Churchill Livingstone, 1985.
- SCOTT DB, OWEN JA, RICHMOND JK. Methaemoglobinaemia due to prilocaine. *Lancet* 1964; **II**: 728-9.
- VICKERS MD, SCHNIEDEN H, WOOD-SMITH FG. *Drugs in anaesthetic practice*, 6th edn. London: Butterworths, 1984.
- COUSINS MJ, BRIDGENBAUGH PO. *Neural blockade in clinical anesthesia and management of pain*. Philadelphia: Lippincott, 1980.

### Intubation with propofol augmented with intravenous lignocaine

D. Mulholland, MB, BCh, FFARCS, FFARCSI, Registrar, R. J. T. Carlisle, MB, BCh, FFARCSI, Consultant, Daisy Hill Hospital, Newry, Co. Down, Northern Ireland.

#### Summary

Sixty patients of ASA grade 1 and aged 18 to 55 years were admitted to a double-blind study. Anaesthesia was induced with propofol 2.5 mg/kg after intravenous pretreatment with lignocaine 1.5 mg/kg or a similar volume of isotonic saline. The quality of subsequent tracheal intubation was graded and the pressor response to tracheal intubation assessed. There were no significant differences between treatment groups.

#### Key words

Anaesthetics, intravenous; propofol, lignocaine.  
Intubation, tracheal; complications.

Depression of laryngeal reflexes has been reported following induction of anaesthesia with propofol.<sup>1</sup> Keaveney *et al.*<sup>2</sup> achieved a 95% success rate using propofol alone for induction and tracheal intubation. De Grood *et al.*<sup>3</sup> augmented propofol with a topical lignocaine spray to allow intubation without muscle relaxants. Yukioka *et al.*<sup>4</sup> showed that intravenous lignocaine effectively suppressed the cough reflex after intubation under deep halothane anaesthesia. The aim of this study was to compare the quality of tracheal intubation after induction of anaesthesia with propofol in patients who received intravenous lignocaine pretreatment with that in patients who received propofol alone.

#### Method

The study was approved by the regional ethics committee. Sixty patients aged between 18 and 55 years and scheduled to undergo elective general or gynaecological surgery which required tracheal intubation were admitted to the study. All were graded as ASA 1. Patients with dental crowns, a past history of drug sensitivity and those assessed as presenting potential difficulties for tracheal intubation were not studied.

The study was conducted in a double-blind fashion. Informed consent was obtained in all cases and the patients were allocated randomly to one of two groups. All patients were premedicated with diazepam 10 mg orally one hour before operation. Blood pressure was recorded automatically (Critikon, Dinamap) in the anaesthetic room and oxygen saturation was measured by pulse oximetry.

A 20-gauge cannula was sited in a large antecubital vein. The lungs were pre-oxygenated. Only one anaesthetist (alternately R.C. and D.M.) was aware of the group to which the patient was allocated. He injected either lignocaine 1.5 mg/kg or an identical volume of 0.9% saline over 30 seconds, timed by a stopwatch. All patients then received propofol 2.5 mg/kg over 20 seconds. After a further 40 seconds, when verbal response and eyelash reflex were lost, tracheal intubation was attempted by the other anaesthetist.

The quality of intubation was graded by the second anaesthetist according to the following scale: excellent;

relaxed jaw, cords abducted, no movement on intubation: good, relaxed jaw, cords abducted, some movement on intubation: unsatisfactory, difficulty in opening mouth, cords moving or adducted, movement on intubation: failure, laryngoscopy impossible, intubation abandoned.

Failed intubation prompted ventilation of the lungs with 100% oxygen for a further 30 seconds before laryngoscopy was re-attempted. If intubation again proved impossible anaesthesia was deepened with volatile agents and a neuromuscular blocker was administered before intubation was attempted again. Successful intubation was followed by administration of an appropriate anaesthetic, and a neuromuscular blocker was given if required.

A decrease in oxygen saturation during induction was treated by ventilation with 100% oxygen through the face-mask. Mean arterial blood pressure was measured immediately before laryngoscopy, and one minute after tracheal intubation.

The ages and weights of patients in the two groups were compared using the unpaired Student's *t*-test. Sex distribution was analysed by the Chi-squared test and the intubation quality with the Mann-Whitney *U*-test. Blood pressure measurements were compared within groups using the paired *t*-test and between groups using the unpaired *t*-test.

#### Results

Two groups of 30 patients each were studied. There were no significant differences between groups in respect of age or weight (Table 1), but there were significantly more

**Table 1.** Mean (SD) age and weight, and sex distribution, of patients who received pretreatment with lignocaine or saline. There were no significant differences.

	Lignocaine	Saline
Age; years	34.4 (10.7)	32.5 (5.5)
Weight; kg	69.4 (10.1)	68.2 (9.8)
M:F	11:19	7:23



**Table 2.** Quality of intubation assessed by anaesthetist in patients who received pretreatment with lignocaine or saline. Figures indicate number of patients. There were no significant differences.

Grade	Lignocaine	Saline
Excellent	14	9
Good	6	7
Unsatisfactory	2	4
Failed	8	10

**Table 3.** Mean (SD) of mean arterial pressures (mmHg) immediately before laryngoscopy and one minute after tracheal intubation in patients who received pretreatment with lignocaine or saline. There were no significant differences.

	Lignocaine	Saline
Before laryngoscopy	95.1 (15.8)	91.5 (10.9)
After intubation	98.2 (14.2)	92.0 (14.7)

female patients in the pretreatment group. Excellent intubation conditions were recorded in 30% of the control group and 46% of those who received lignocaine pretreatment; this difference was not statistically significant (Table 2). None of the patients in whom tracheal intubation was unsuccessful at the first attempt could be intubated after a 30-second delay. There were no significant differences between the pre-intubation and post-intubation blood pressures in either group (Table 3), and no significant differences in blood pressures between groups.

### Discussion

Previous studies have shown that propofol obtunds laryngeal reflexes and that laryngoscopy and intubation are possible at safe induction doses.<sup>1,2</sup> Lignocaine is known to reduce tracheal irritability when injected intravenously and seems a logical pretreatment when propofol is used in this way.<sup>4</sup> Keaveney *et al.*<sup>2</sup> used the same induction dose as ourselves but slightly different intubation criteria; they reported that intubating conditions were ideal in 60% of patients.

Yukioka *et al.*<sup>4</sup> reported a dose-dependent suppression of coughing by intravenous lignocaine. However, in a pilot study we found that doses greater than 1.5 mg/kg caused a tingling sensation which would have defeated the double-blind design of our study.

The hypertensive response to laryngoscopy and intubation was not evident in either group. Intravenous lignocaine pretreatment has been recommended for the control of this reflex following induction with thiopentone but did not seem to offer any advantage in this study. Thus an induction dose of propofol 2.5 mg/kg seems to result in cardiovascular stability during subsequent laryngoscopy and intubation although we measured blood pressure on only two occasions. This effect was evident in patients in whom intubating conditions were unsatisfactory or in whom intubation was unsuccessful. It appears that movement and coughing during intubation after induction with propofol is not related to an increase in blood pressure.

This technique may have a place in the induction and intubation of patients in whom nondepolarising muscle relaxants are contraindicated (for example patients with myasthenia gravis) or when the anaesthetist wishes to avoid suxamethonium. It appears to offer cardiovascular stability regardless of the amount of movement during intubation. However, it does not seem reliable or predictable enough for routine use as there was an overall failure rate of 30.6%.

### References

1. McKEATING K, BALI IM, DUNDEE JW. The effects of thiopentone and propofol on upper airway integrity. *Anaesthesia* 1988; **43**: 638–40.
2. KEAVENEY JP, KNELL PJ. Intubation under induction doses of propofol. *Anaesthesia* 1988; **43** (Suppl.): 80–1.
3. DE GROOD PMRM, VAN EGMOND J, WETERING M VAN DE, VAN BEEM HB, BOOU LHDJ, CRUL JF. Lack of effects of emulsified propofol ('Diprivan') on vecuronium pharmacodynamics—preliminary results in man. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 28–30.
4. YUKIOKA H, YOSHIMOTO N, NISHIMURA K, FUJIMORI M. Intravenous lidocaine as a suppressant of coughing during tracheal intubation. *Anesthesia and Analgesia* 1985; **64**: 1189–92.
5. HAMILL JF, BEDFORD RF, WEAVER DC, COLOHAN AR. Lidocaine before endotracheal intubation: intravenous or laryngotracheal? *Anesthesiology* 1981; **55**: 578–81.

## Correspondence

<b>Dosage of phenylephrine in spinal anaesthesia for Caesarean section</b>	314	<b>Hypotension and brain death?</b>	324
<i>J.C. Taylor, FFARCS and M. E. Tunstall, FFARCS</i>		<i>N.W. Goodman, FFARCS</i>	
<b>Hazard: single-use parallel Lack breathing system</b>	316	<i>J.M. Leigh, FFARCS</i>	325
<i>P.L. Jones, FFARCS</i>		<b>Failure of the Ruben circle control valve</b>	325
<i>S.K. Williams</i>	317	<i>M.R. Logan, FFARCS</i>	
<b>Anaesthesia for laparoscopic cholecystectomy</b>	317	<i>J. Syders</i>	326
<i>J.M. Stanton, FFARCS</i>		<b>Another complication of arterial cannulation</b>	326
<i>A.C. Greville, MB, BS and E.A.F. Clements, FFARCS</i>	317	<i>M.M. Brown, BM, BS</i>	
<b>Other implications of laser laparoscopic cholecystectomy</b>	318	<b>Acute renal failure in association with a pneumatic antishock garment and with tense ascites</b>	326
<i>P. Joshi, FFARCS, A. Mahoney, FFARCS and N.C. Soni, FFARCS</i>		<i>M.J. O'Leary, MB and G.R. Park, FFARCS</i>	
<b>The Level 1 Fluid Warmer</b>	318	<b>Cricoid pressure in the sniffing position</b>	327
<i>J.P. Nolan, FCAnaes and A.A.C. Dow, FCAnaes</i>		<i>A.D. Baxter, FFARCS</i>	
<b>Blood or fluid warmers?</b>	319	<b>A complication of a flexible tracheostomy tube during laryngectomy</b>	328
<i>R.G. Presson, Jr MD, S.C. Hillier, FFARCS and K.A. Haselby, MD</i>		<i>T. Ezri, MD, D. Halperin, MD, S. Lurie, MD and D. Soroker, MD</i>	
<b>Novel use of the Rendell–Baker Soucek mask</b>	319	<b>Sexual excitement following anaesthesia or sedation</b>	328
<i>D. Northwood, FFARCS and M.J. Wade, FFARCS</i>		<i>B.J. Woodcock, MRCP, FCAnaes</i>	
<b>Anaesthesia in the field</b>	319	<b>The cost of propofol infusion in neurosurgery</b>	329
<i>S.Q.M. Tighe, FFARCS</i>		<i>H.A. Aitken, FFARCSI and P.A. Farling, FFARCSI</i>	
<i>J. Restall, FFARCS and I.G. Johnston, FFARCSI</i>	320	<b>Regional anaesthesia must be properly managed</b>	329
<b>The laryngeal mask airway in paediatric anaesthesia</b>	321	<i>R.H. Dale, MD</i>	
<i>D.E. Withington, MRCP, FFARCS</i>		<b>Suxamethonium and intensive care</b>	330
<i>S.R. Wrigley, FCAnaes, H.E. Jones, FCAnaes, D.F. Johnston, FRCS and P.J. Robb, FRCS</i>	321	<i>J. Coakley, MRCP</i>	
<b>Exploding ampoules</b>	322	<b>Insertion of laryngeal mask airway in children</b>	330
<i>S. Williams, FFARCS</i>		<i>L.R. McNicol, FFARCS</i>	
<i>A.M. Cotterell</i>	322	<b>Race and Apgar scores</b>	330
<b>Disposable carbon dioxide detectors</b>	323	<i>A.Z. Yama, MD and G.F. Marx, MD</i>	
<i>J.D. Muir, MB, ChB, P.B. Randalls, FFARCS, G.B. Smith, FFARCS and B.L. Taylor, FFARCS</i>		<b>The Rex Binning Department of Anaesthesia</b>	331
<i>D.J. Wilkinson, FFARCS, W.T. Denman, FCAnaes, D. Higgins, FCAnaes, M. Hayes, FCAnaes and E. Yau</i>	324	<i>P.A.D. Williams, FFARCS</i>	
<i>D. O'Flaherty and A.P. Adams</i>	324	<b>Nudging the emergency oxygen</b>	331
		<i>Z. Hanafiah, FFARCSI and W.F.S. Sellers, FFARCS</i>	

### Dosage of phenylephrine in spinal anaesthesia for Caesarean section

Phenylephrine, an  $\alpha_1$ -selective agonist, has been re-introduced as a vasopressor for the treatment of hypotension caused by spinal anaesthesia for Caesarean section.<sup>1,2</sup> In our experience the role of this agent is invaluable. Apart from a few published articles,<sup>1,2</sup> there is no guidance for use in obstetrics on dosage or method of use either in the *British National Formulary*,<sup>3</sup> in 'Goodman and Gilman',<sup>4</sup> or other commonly consulted sources.<sup>5–7</sup> We wish to report two cases of phenylephrine overdose in our Maternity Unit which were the result of individuals hearing about the use

of phenylephrine but who were not aware of the doses used. They therefore consulted the manufacturers data sheet for guidance. Its recommended minimum intravenous dose of phenylephrine is 500  $\mu\text{g}$ . They recommend 5000  $\mu\text{g}$  by intramuscular injection for hypotension during spinal anaesthesia. It has to be stated that the manufacturers are unable to give guidance on the obstetric use of phenylephrine until there is more information available and until the product licence is changed. There is no mention of pregnancy in the current data sheet.

All correspondence should be addressed to Dr M. Morgan, Editor of *Anaesthesia*, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, United Kingdom.

Letters must be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters contained in the Notice to Contributors to *Anaesthesia* printed at the back of each issue. The degrees and diplomas of each author must be given in a covering letter personally signed by all the authors.

Correspondence presented in any other style or format may be the subject of considerable delay and may be returned to the author for revision. *If the letter comments on a published article in Anaesthesia, please send three copies; otherwise two copies of your letter will suffice.*

The first patient was a 33-year-old, para one, in labour who required a Caesarean section. She chose to be awake under spinal anaesthesia. Her circulation was preloaded with 1 litre of saline. Ephedrine 30 mg was given intramuscularly after the spinal injection of 2.5 ml of hyperbaric 0.5% bupivacaine. Her blood pressure decreased after induction to 95 mmHg systolic, the pulse rate increased to 150/minute and she felt faint. She was tilted further over to the left and a 5–10° head-down table tilt was applied. Her low blood pressure, tachycardia and other symptoms persisted. She was given phenylephrine 750 µg intravenously. Almost immediately this produced a pulse rate of 35/minute and there were multiple ventricular ectopics. She developed an extremely severe headache and slightly blurred vision. Her blood pressure at this point was 250/140 mmHg. She was given hydralazine 20 mg intravenously and then phentolamine 5+5 mg intravenously. The headache subsided when the systolic blood pressure decreased to below 180 mmHg. The headache persisted in a mild form, together with blurred vision for a further 6 hours. The fetal heart rate was checked frequently during the whole of this episode and was found to be undisturbed, and upon delivery 15 minutes later, the baby cried quickly and attained Apgar-minus-colour scores of 8 and 8 at 1 and 5 minutes respectively. The mother was examined by a consultant neurologist 3 days later and was found to be normal and unaffected.

The second patient, a 30-year-old para 0 in labour required a rotational forceps delivery for deep transverse arrest. The blood pressure decreased after induction of spinal anaesthesia to 101/59 mmHg and there were symptoms of faintness and nausea. She was given phenylephrine 250 µg intravenously. This was followed by a severe occipital headache coinciding with a blood pressure increase to 183/91 mmHg. There was a decrease in the fetal heart rate (FHR) associated with the peak of blood pressure. The baby was delivered 8 minutes after the FHR nadir of 65 beats/minutes. The Apgar-minus-colour score was 6 and 8 at 1 and 5 minutes respectively.

It was considered at one time that the prophylactic or therapeutic use of vasopressor agents in conjunction with spinal or caudal anaesthesia during pregnancy was contraindicated.<sup>8</sup> This view was based on the results of experiments in sheep where the adverse effect of vasopressors on uterine blood flow was demonstrated. In the case of phenylephrine the dosage in the sheep was greatly in excess of that currently recommended for the treatment of hypotension due to spinal anaesthesia in obstetrics.<sup>1,2</sup> Investigations in uterine artery blood flow in 1974 demonstrated that ephedrine would be the vasopressor of choice in obstetrics.<sup>9</sup> Various vasopressor agents were administered to chronically implanted standing nonanaesthetised pregnant sheep. In the investigations of metaraminol<sup>9</sup> the lowest dose used was 50–100 times that which would be required to restore the blood pressure in a hypotensive pregnant human, under spinal anaesthesia, with left uterine displacement and with a 1 litre fluid preload before induction.

Short periods of maternal hypotension (< 2 minutes) are not harmful to the neonate.<sup>10</sup> It is advisable, nevertheless, to administer a vasopressor intravenously to the mother as soon as her blood pressure begins to decrease. This policy often prevents hypotension (a decrease in systolic pressure either by more than 30 mmHg or to below 100 mmHg). It should also prevent the mother from experiencing faintness or nausea, apart from being of benefit to the fetus.

Ephedrine is the vasopressor recommended in all current texts on obstetric anaesthesia, but Moir and Thorburn have stated that current therapy for hypotension in the extensively sympathetically blocked patient is not entirely

satisfactory.<sup>11</sup> Ramanathan has recommended phenylephrine injection if hypotension remains unresponsive to ephedrine administration.<sup>12</sup> The occasional obstetric patient requires a dosage of ephedrine to restore the blood pressure which produces unpleasant side effects such as a thumping heart and a pulsating headache.

Ephedrine is both an  $\alpha$ - and a  $\beta$ -adrenoceptor agonist. It also enhances the release of noradrenaline from sympathetic neurones. The drug stimulates heart rate and cardiac output and variably increases peripheral resistance.<sup>4</sup> The pressor responses to ephedrine are mainly due to cardiac stimulation. Phenylephrine is an  $\alpha_1$ -selective agonist. It activates  $\beta$ -adrenoceptors only at much higher concentrations.<sup>4</sup>

The occasional inadequacy of ephedrine for the restoration of maternal blood pressure and relief of symptoms accounts for the interest that is now being taken in phenylephrine. Our experience with over 100 mothers for Caesarean section under spinal anaesthesia who require a vasopressor confirms that intravenous bolus doses of phenylephrine are consistently and promptly effective in the relief of maternal hypotension and its associated symptoms.

The recommended dosage range is 20–100 µg by intravenous bolus.<sup>1,2</sup> If intravenous ephedrine 5–10 mg has been used and found to be unsatisfactory, phenylephrine 20–40 µg may be safely superimposed to produce the required effect.<sup>1</sup> The intravenous bolus dose may be repeated or increased (maximum 100 µg per bolus) as required. It is uncommon for cumulative intravenous bolus dosage of phenylephrine to exceed 200 µg. The largest cumulative dosage we have recorded to date is 480 µg, and the duration of action of an effective intravenous bolus dose is 2–5 minutes.

It goes without saying that no pregnant patient under spinal anaesthesia should be administered a vasopressor without attention being given before to adequate relief of caval compression and enhancement of venous return. In the 20 minutes before spinal anaesthesia the mother should have also received a one litre intravenous preload with saline or a balanced electrolyte solution. The most convenient way to prepare the phenylephrine for use is to add the contents of the 10-mg ampoule to 500 ml saline or similar. This gives 20 µg/ml. The solution is then drawn up in a labelled 10-ml syringe. The effectiveness of phenylephrine is very reassuring and it would now be difficult to imagine conducting a spinal anaesthetic for Caesarean section without it being available. A safe secure and certain control of maternal blood pressure is one of many requirements for ensuring 'the perfect experience' for the mother having her baby by Caesarean section under regional blockade.

Aberdeen Royal Infirmary,  
Foresterhill,  
Aberdeen AB9 2ZB

J.C. TAYLOR  
M.E. TUNSTALL

## References

1. RAMANATHAN S, GRANT GJ. Vasopressor therapy for hypotension due to epidural anesthesia for Cesarean section. *Acta Anaesthesiologica Scandinavica* 1988; 32: 559–65.
2. MORAN DH, PERILLO M, BADER AM, DATTA S. Phenylephrine in treating maternal hypotension secondary to spinal anesthesia. *Anesthesiology* 1989; 71: A 857.
3. Joint Formulary Committee of the British Medical Association and the Royal Pharmaceutical Society of Great Britain. *British National Formulary*, No. 20: London: British Medical Association, Pharmaceutical Press, 1990: 89.
4. GILMAN AG, RALL TW, NIES AS, TAYLOR P. *The pharmacological basis of therapeutics*, 8th edn. New York: Pergamon Press, 1990: 207 and 213.

5. REYNOLDS, JRF, ed. *Martindale, the extra pharmacopoeia*, 29th edn. London: The Pharmaceutical Press, 1989: 1473-5.
6. VICKERS MD, SCHNEIDEN H, WOOD-SMITH FG. *Drugs in anaesthetic practice* 6th edn. London: Butterworths, 1986.
7. CALVEY TN, WILLIAMS NE. *Principles and practice of pharmacology for anaesthetists*. Oxford: Blackwell Scientific, 1982.
8. GREISS FC, CRANDELL DL. Therapy for hypotension induced by spinal anesthesia during pregnancy. *Journal of the American Medical Association* 1965; **191**: 793-6.
9. RALSTON DH, SHNIDER SM, DE LORIMIER AA. Effects of equipotent ephedrine, metaraminol, mephentermine, and methoxamine on uterine blood flow in the pregnant ewe. *Anesthesiology* 1974; **40**: 354-70.
10. CORKE BC, DATTA S, OSTHEIMER GW, WEISS, JB, ALPER MH. Spinal anaesthesia for Caesarean section: the influence of hypotension on neonatal outcome. *Anaesthesia* 1982; **37**: 658-62.
11. MOIR DD, THORBURN J. *Obstetric anaesthesia and analgesia*, 3rd edn. London: Bailliere Tindall, 1986: 220.
12. RAMANATHAN S. *Obstetric anaesthesia*. Philadelphia: Lea and Febiger, 1988: 101.

### Hazard: single-use parallel Lack breathing system

The latest introduction to the excellent range of single-use breathing systems from Intersurgical Ltd constitutes a serious potential hazard. Figure 1 illustrates the conventional configuration of the components in the product. It departs from a Mapleson 'A' or Magill system only because the unidirectional spill APL valve has been extended by a flexible hose to return it to a position of convenience adjacent to the reservoir bag. The bag is retained on a 22 mm detachable taper. This is conventional and acceptable practice. The APL valve is also retained on a 22-mm taper (Fig. 2). This is neither conventional nor safe. It permits the bag and the valve to be separated from the tubing system and transposed (Fig. 3). The patient, so connected, is then separated from fresh gas flow by a metre of tubing, a deadspace configuration that is likely to be fatal.

One promotional sample of this product was delivered into a clinical area and fell into naive hands. Fortunately, although misassembly occurred, the product was retrieved before it was used on a patient. Since its discovery, the company has acted promptly to withdraw all known examples of the product and has, following clinical discussions, offered a prototype of proposed future production in which the APL valve is permanently bonded to the main system assembly, thereby eliminating the hazard.

It is essential that the profession enters into discussion with suppliers to establish a code of practice for company representatives which prevents their distributing samples to anyone other than professionally authorised users. At its

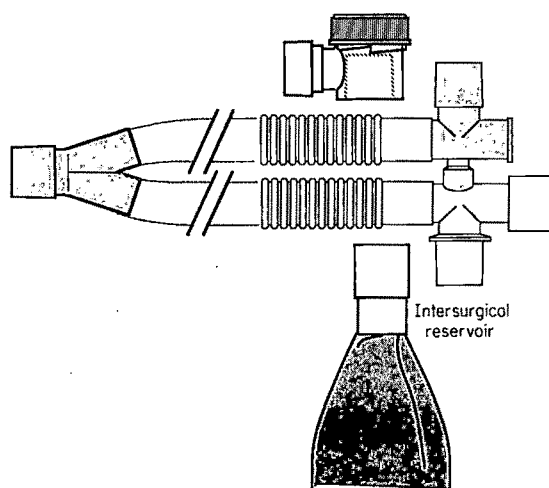


Fig. 2. Intersurgical parallel Lack breathing system with components separated.

lowest level, this should mean that such product is introduced into the clinical environment only upon the specific instruction of a consultant anaesthetist who accepts responsibility for ensuring that all relevant staff have been trained in its use.<sup>1</sup> If we do not exercise such restraint as assures Health Authorities that we can control the quality

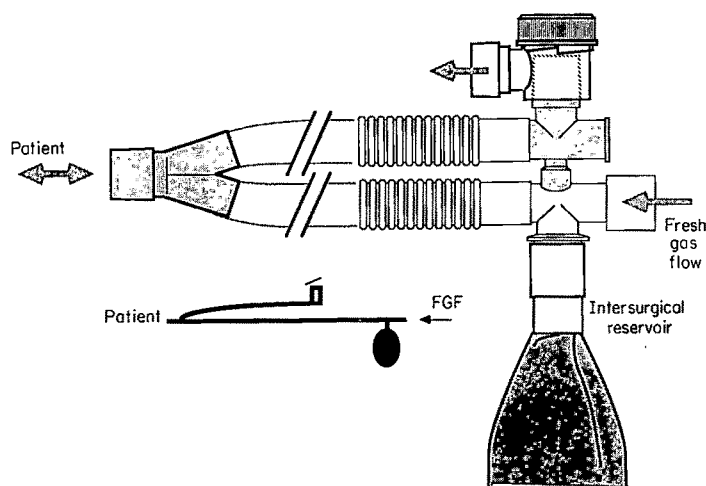


Fig. 1. Intersurgical parallel Lack breathing system. Normal assembly of components to perform as a Mapleson 'A' system.

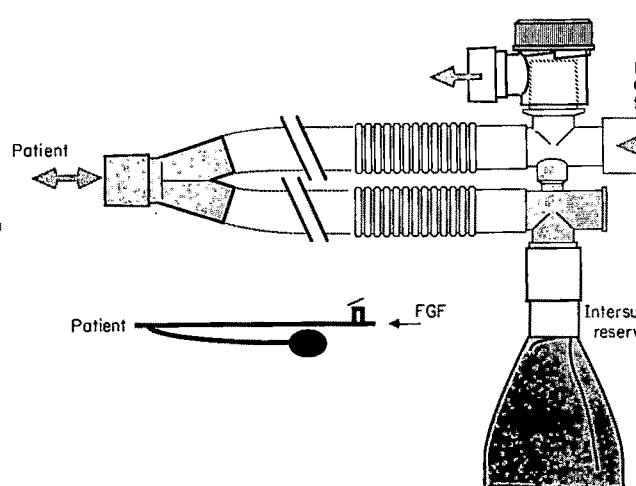


Fig. 3. Intersurgical parallel Lack breathing system with components wrongly assembled to cause massive rebreathing.

0/0

## I.V. FLUIDS

## BLOOD PRESSURE

TIME :

B.P.

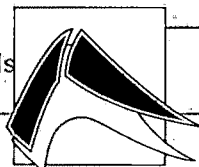
Heart  
rate

Temp

190  
180  
170  
160  
150  
140  
130  
120  
110  
100  
90  
80  
70  
60  
50  
40  
30  
20  
10  
0

**POTENT  
PLASMA  
VOLUME  
EXPANSION  
FOR AROUND  
12 HOURS**

I.V.  
Fluids



# PENTASPAN®

Pentastarch 10%

## More than just a replacement fluid

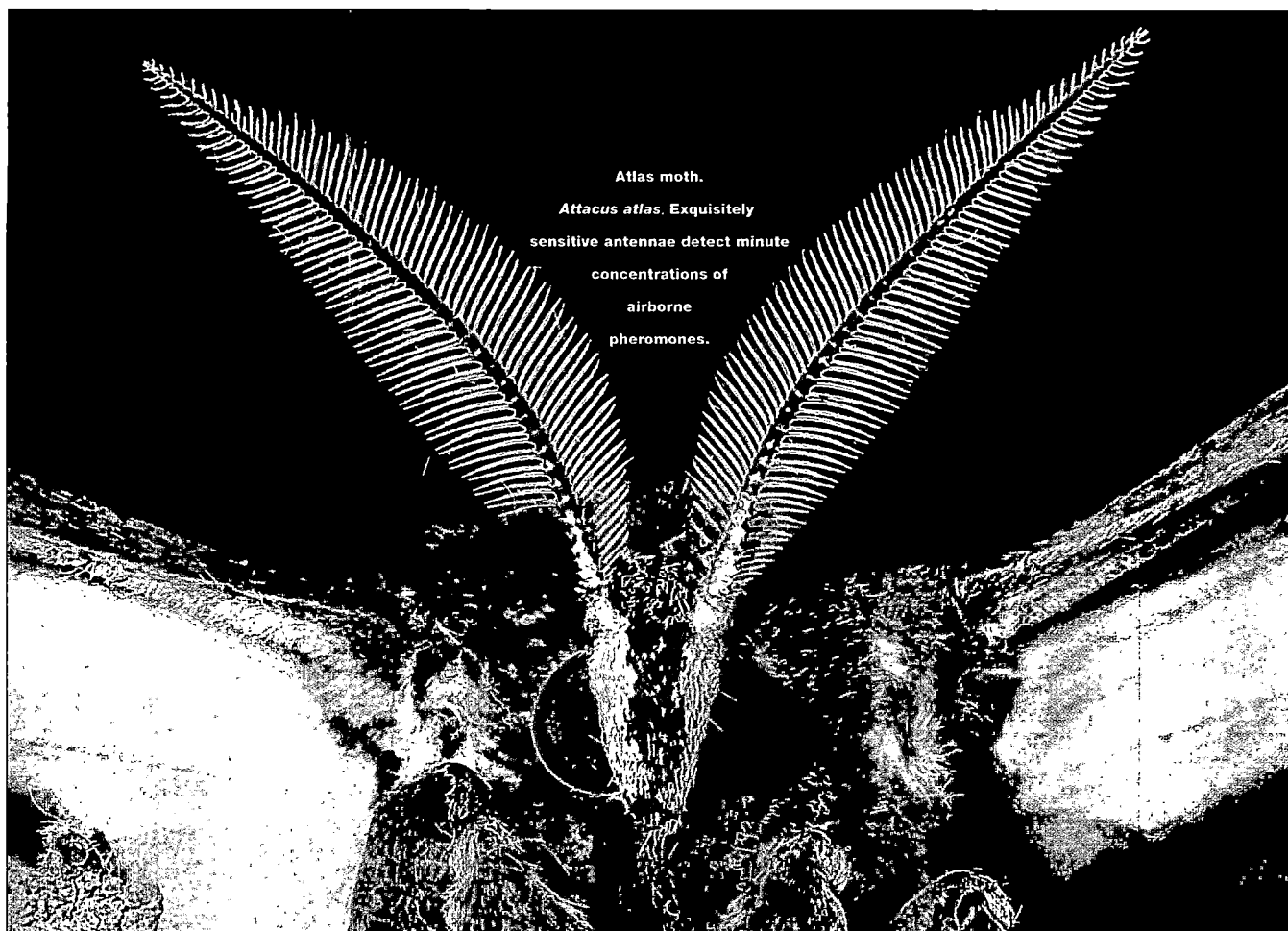
**Pentaspan® ▼ Abbreviated Prescribing Information (10% pentastarch) Presentation 250 and 500 ml PVC bags containing 10% pentastarch in 0.9% saline.** It is a sterile, non-pyrogenic, colloidal solution for intravenous infusion only. **Uses** As a plasma volume expander in cases of hypovolaemia, and leucapheresis. **Dosage and Administration** Plasma volume expansion: Intravenous infusion only. Total dosage and rate of infusion depend upon the amount of blood or plasma lost. In adults the amount usually administered is 500-2000 ml. Total daily dosage does not usually exceed 2000 ml for a typical 70 kg patient or 28 ml per kg of body weight. In acute haemorrhagic shock an administration rate approaching 20 mg/kg/hr may be used. Use beyond 72 hours has not been studied. Leucapheresis: 250 to 700 ml Pentaspan injection to which citrate anticoagulant has been added is typically administered by the aseptic addition to the input line of the centrifugation apparatus at a ratio of 1:8 to 1:13 to venous whole blood. The bottle containing Pentaspan and citrate should be thoroughly mixed to assure effective anticoagulation of blood as it flows through the leucapheresis machine. The safety and compatibility of other additives have not been established. **Contraindications, Precautions and Warnings** Contraindicated in patients with known hypersensitivity to hydroxyethyl starch, with severe bleeding disorders or with severe congestive heart failure; with renal failure with oliguria or anuria not related to hypovolaemia; and with acute cerebral infarction where cerebral oedema is a potential problem. Haematocrit and haemoglobin may be decreased and plasma proteins diluted excessively by administration of large volumes of Pentaspan. Large volumes of Pentaspan may slightly alter the coagulation mechanism. Isovolemic substitution with Pentaspan up to 20% does not interfere with cross-matching determinations. Higher dilutions may lead to rouleaux formation. Pentaspan should be given

with primary renal impairment with a history of liver disease. There is no evidence from clinical trials that Pentaspan is antigenic. Pentaspan has been reported to produce sensitivity reactions such as wheezing and urticaria. If such reactions occur, they are readily controlled by discontinuation of Pentaspan and, if necessary, administration of an antihistaminic agent. Elevated serum amylase levels may be observed temporarily following administration of Pentaspan although no association with pancreatitis has been demonstrated. Pentaspan is not suitable as a replacement for the fresh components of plasma or the cellular components of blood. Regular and frequent clinical evaluation including complete blood counts (CBC) are necessary for monitoring during leucapheresis using Pentaspan. **Use in pregnancy** High doses (40 ml/kg/day) of Pentaspan increased the number of resorptions and minor visceral abnormalities in rabbits and reduced nidation in mice. It should not be used, particularly during early pregnancy, unless in the judgement of the physician the potential benefits outweigh the potential hazards. It is not known whether Pentaspan is excreted in human milk therefore caution should be exercised in administering it to nursing mothers. **Use in children** The safety and efficacy of Pentaspan in children have not been established. **Adverse Effects** Coagulation disorders have been reported. **Product Licence Holder and Number** Du Pont (U.K.) Ltd PL 4524/0038 **Basic NHS Price** 250 ml x 10 £79.50 500 ml x 10 £157.00 Data sheet with full prescribing information is available on request from Du Pont Pharmaceuticals Limited, Wedgwood Way, Stevenage, Hertfordshire SG1 4QN. **Date of Preparation** October 1990

**DU PONT**

® Du Pont Trademark

DU PONT  
PHARMA



Atlas moth.  
*Attacus atlas*. Exquisitely  
 sensitive antennae detect minute  
 concentrations of  
 airborne  
 pheromones.

# Is this an anaesthetist's role model?

*An anaesthetist, like a moth,  
 develops a sensitivity bordering on "sixth sense";  
 mental antennae attuned to every  
 requirement of the patient.*

**Diprivan**<sup>▼</sup>  
 propofol  
 i.v. anaesthesia

## P R E S C R I B I N G   N O T E S

▼Special reporting to the CSM required.

Consult data sheet before prescribing.

**Use:** Induction and maintenance of general anaesthesia for surgical procedures lasting up to one hour.

**Presentation:** Ready to use, isotonic, aqueous emulsion containing 10mg/ml propofol in a vehicle containing soybean oil and purified egg phosphatide.

**Dosage and administration:** **Induction:** Titrate against response using approximately 4ml every 10 seconds in healthy adults and 2ml every 10 seconds in patients of ASA grades 3 and 4. Patients under 55 years are likely to require 2.0 to 2.5mg/kg, older patients may require approximately 20% less.

**Maintenance:** Usually 0.1 to 0.2mg/kg/min (6 to 12mg/kg/hr). Continuous infusion may require slightly higher rates for 10 to 20 minutes after induction. The infusion can be used undiluted or diluted with 5% Dextrose (intravenous infusion BP). Alternatively repeat bolus injections of 25 to 50mg (2.5 to 5.0ml) may be administered according to clinical need.



**Paediatric Use:** No experience in children or in mothers who are breast feeding.

**Contraindications:** Allergy to 'Diprivan'.

**Precautions:** Ampoules and vials should be inspected before use for particulate matter and discolouration. Aseptic technique should be followed throughout its handling; administration commencing without delay once drawn into the syringe or giving set. Discard any unused 'Diprivan' after single patient use. Do not mix prior to administration with other agents or infusion fluids other than Dextrose 5%, such dilutions should be prepared immediately before administration. Hypotension and transient apnoea may occur during induction. Occasionally, hypotension may require use of i.v. fluids and a lower rate of administration during maintenance. Apply caution in cardiac, respiratory, renal or hepatic impairment; epilepsy; in hypovolaemic or debilitated patients; and in disorders of fat metabolism or conditions where lipid emulsions should be used cautiously. Bradycardia may occur due to the lack of vagolytic activity of 'Diprivan' and administration of an anticholinergic should be considered particularly if vagal

tone is dominant. Do not use in pregnancy except for termination. Discharge after general anaesthesia - allow adequate time for full recovery.

**Side effects:** Epileptiform movements, including convulsions and opisthotonus, have occurred rarely in a temporal relationship to 'Diprivan'. Nausea, vomiting and headache in a small proportion of patients. Very rarely, clinical features of anaphylaxis, which may include bronchospasm and erythema accompanied by hypotension, have been reported with 'Diprivan'. Pain on injection in a proportion of patients. Discolouration of urine, venous sequelae and fever are rare. Minimal evidence of excitation on induction.

**Product licence number:** (29/0190). **Basic NHS cost:** £3.98 per 20ml ampoule, £9.95 per 50ml infusion vial.

'Diprivan' is a trademark.

Further information is available from ICI Pharmaceuticals (UK), Southbank, Alderley Park, Macclesfield, Cheshire SK10 4TF.



and safety of anaesthesia, the clinical freedom of the profession will be justifiably suppressed by administrative intervention.

*University Hospital of Wales,  
Cardiff CF4 4XW*

P.L. JONES

#### Reference

1. Hospital Equipment Information No. 98: 'Management of Equipment'. Department of Health, First published January 1982, Section 3, 8.

#### A reply

Intersurgical are grateful to Dr P. Jones for bringing this potential problem to our attention. After invaluable discussions with Dr Jones, we can confirm that the above-mentioned modifications have been made to this product, therefore eliminating this potential problem. Intersurgical entirely support the thoughts of Dr Jones that the profession should enter into discussion with suppliers to ensure that the correct and safe design of product reaches the clinical environment.

*Crane House, Gould Road,  
Twickenham, Middx TW2 6RS.*

S.K. WILLIAMS

### Anaesthesia for laparoscopic cholecystectomy

The recently published case report of anaesthesia for laparoscopic cholecystectomy (*Anaesthesia* 1990; 45: 944-5) illustrates the erroneous impression that can be gained from a single case report. It certainly does not reflect the experience, still limited, in this district general hospital.

Since July 1990, 29 elective laparoscopic cholecystectomies have been performed at this hospital. The patients were between 22 and 72 years of age, ASA1 to 3; 27 patients were discharged one to three days after operation. One patient remained in hospital for 4 postoperative days because of unstable diabetes mellitus complicated by vomiting, and another for 10 days because of a subsequent laparotomy for bleeding. Operating time, not including anaesthetic time, varied from 22 to 180 (mean 75) minutes. The operation is performed with normal lighting levels. The excellent screen and camera ensure that it is not necessary to operate in a darkened operating theatre. The size of pneumoperitoneum is larger for upper abdominal than lower abdominal laparoscopy, but we did not need to change the carbon dioxide cylinder until we were operating on the 21st patient.

Initially the most frequent postoperative complication was the high incidence of nausea and vomiting (42% and 42% respectively). At this time we were using a similar technique to that used by Greville and Clements. The anaesthetic technique has subsequently been modified as follows: the peri-operative use of opioids has been reduced by using an intra-operative interpleural bupivacaine block and postoperative intramuscular diclofenac; volatile agents have been abandoned and replaced by a propofol infusion; domperidone is used pre- and postoperatively and a nasogastric tube is used to ensure the stomach is free of bile refluxing from the duodenum. Seven patients have been anaesthetised with this technique; one has vomited once and two have felt nauseated. This has not reached the level of statistical significance, but is a trend in the right direction.

The implications for the anaesthetic department in this district general hospital are at the moment those of providing anaesthetic services in an isolated operating theatre which has been used solely for endoscopy over the last 5 years. All the equipment has to be moved from the main operating theatre to this unit for the all-day list of laparoscopic cholecystectomies.

The Nd:Yag laser is not used for laparoscopic cholecystectomies at the moment, but we are fully equipped with it for use during gastroscopy. If we were to use the laser we would be using a resource already available. The

only capital expense the hospital has incurred is the provision of instruments, carbon dioxide insufflation and camera (approximately £12 000).

I would be interested to hear details of the experience of other anaesthetists, especially if they have noticed the high incidence of postoperative nausea and vomiting and found a solution. I think our experience shows that it is reasonable to have an optimistic outlook as regards the implications of anaesthesia for laparoscopic cholecystectomies in an average district general hospital.

*Airedale General Hospital,  
Keighley,  
West Yorkshire BD20 6TD*

J.M. STANTON

#### A reply

Thank you for giving us the opportunity to comment on Dr Stanton's experience.

We are sorry that our case report (*Anaesthesia* 1990; 45: 944-5) gives an erroneous impression. It was written in order to document laparoscopic laser cholecystectomy in the anaesthetic literature and to discuss the impact of the introduction of a new operation using new technology on a busy district general hospital. We firmly believe that this operation represents a major advance in minimal invasive surgery with a high level of patient acceptance. We appreciate that laparoscopic cholecystectomy is performed in many centres using diathermy rather than Nd:Yag laser.

It is of interest that the laser is not used for this operation at Airedale General Hospital, despite its availability. We believe that there are a number of advantages of the laser over diathermy: minimal smoke production and no interference with the televisual relay, thus improving operative conditions; extremely precise cutting and reduced tissue trauma, possibly with less postoperative pain.

We have now performed 65 cases (58 with laser alone, five with laser and diathermy, and two with diathermy alone) and there have been several modifications to the documented anaesthetic technique. Postoperative nausea and vomiting have not been a serious problem. We are currently evaluating our results.

*Whipps Cross Hospital,  
London E11 1NR*

A.C. GREVILLE  
E.A.F. CLEMENTS

### Other implications of laser laparoscopic cholecystectomy

We read with interest the article by Greville and Clements (*Anaesthesia* 1990; 45: 944-5). Our establishment is also undertaking laser laparoscopic cholecystectomies and we would like to highlight two of the problems that we have encountered.

A 65-year-old, 75 kg woman, otherwise fit and healthy, presented for routine laser laparoscopic cholecystectomy. The procedure lasted 5 hours and was complicated by bleeding from the cystic artery. The intra-operative blood loss was 3200 ml which necessitated a laparotomy to achieve haemostasis. The patient was admitted to the intensive care unit for postoperative management. She was transferred back to the general ward 24 hours later.

The second patient was a 46-year-old, 85 kg man, a known smoker and also suffering from a hiatus hernia and diabetes mellitus. On examination there were no abnormal physical findings, he had good neck movements and mouth opening (Mallampati class II). Premedication included prophylactic antacids. After pre-oxygenation, thiopentone and suxamethonium were given and cricoid pressure applied. At laryngoscopy only the tip of the epiglottis could be visualised. Tracheal intubation was unsuccessful using various manoeuvres. Cricoid pressure was maintained and the patient allowed to breathe spontaneously. Under deep halothane anaesthesia the trachea was successfully intubated, although with some difficulty. Despite these problems the laparoscopic cholecystectomy proceeded as planned. It lasted 5 hours and the patient's trachea was extubated when he was awake. However, 30 minutes later it became obvious that he was bleeding. The systolic blood pressure was 80 mmHg, heart rate 140 beats/minute and

his abdomen was significantly distended. He was resuscitated with a rapid infusion of colloid and blood, despite which it was difficult to maintain his blood pressure. An urgent laparotomy was required. The patient at this time was confused, restless and uncooperative. The options were limited, but awake fiberoptic intubation was determined to be the most suitable technique. The trachea was successfully intubated and at laparotomy the intraperitoneal blood loss was measured at 4000 ml. Haemostasis was achieved by tying off an 'aberrant' artery. He was transferred to the intensive care unit for postoperative management, and discharged from the unit 24 hours later.

These two cases bring to attention the potentially prolonged duration of this expanding branch of surgery. There is also a risk of major haemorrhage which may be missed intra-operatively. It is obvious that anaesthetic problems such as difficult intubation should preclude selection of patients from a relatively new surgical procedure. With hindsight we should have abandoned the planned surgery and proceeded to conventional surgery.

In conclusion, we would recommend that 'experimental' surgery be limited to ASA 1 or 2 patients without anaesthetic complications. All other patients should undergo alternative conventional surgery which is known to have a low morbidity.

Westminster Hospital,  
London SW1P 2AP

P. JOSHI  
A. MAHONEY  
N.C. SONI

### The Level 1 Fluid Warmer

We were most interested in the evaluation of the Level 1 blood warmer by Drs Browne, De Boeck and Morgan (*Anaesthesia* 1990; 45: 960-3). We have been working in the Maryland Institute of Emergency Medical Services Systems (MIEMSS) Shock Trauma Center and would like to make the following comments, based on our personal clinical experience (approximately 100 severe trauma cases) with the Level 1 System 500 Fluid Warmer (Level 1 Technologies, Inc. Marshfield, MA, USA).

The system can always be set up and primed easily and rapidly. Indeed, since the disposable set includes an air eliminator it is consistently easier to prime than a standard blood administration set. Each of the six Level 1 warmers used at MIEMSS is fitted with two Alton Dean pressure infusers (Alton Dean Medical, Inc, Utah, USA).<sup>1</sup> Fluid bags in the infusers can be changed very rapidly and a simple on-off switch produces immediate inflation to the desired pressure. We have found that the combination of the two or three spike giving sets and the automatic pressure infusers simplifies the whole process of rapid blood transfusion.

On most occasions we use the Level 1 warmer in combination with an 8.5-gauge cannula (pulmonary artery catheter introducer sheath), placed in the femoral, internal jugular or subclavian vein. This permits maximum utilisation of the wide-bore (4.8 mm ID) giving set and results in significantly faster flow rates in comparison with a 14-gauge cannula.<sup>2</sup>

Severe hypothermia is common among severely injured patients<sup>3</sup> and massive blood transfusion will usually compound the problem. Under these circumstances, use of

the Level 1 warmer during transfusion will maintain, and often increase, the core temperature.

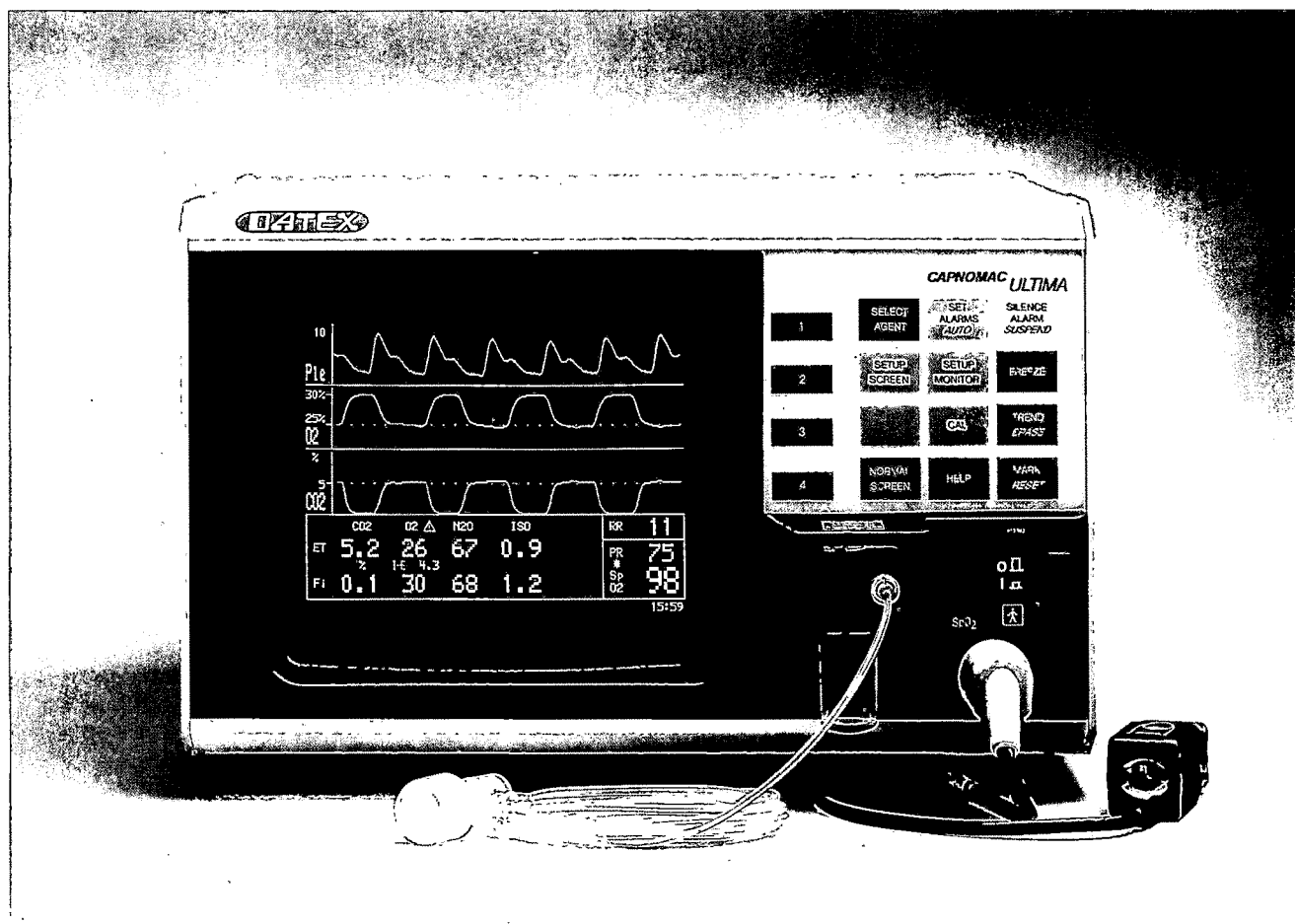
In our experience, we can report only one problem with the Level 1 warmer. The 170- $\mu$ m screen filter, situated distal to the heat exchanger, has a tendency to become obstructed with blood clot. How quickly this occurs depends on the age of the transfused blood, but on a few occasions the filter has become totally blocked after administering just six units. The in-line filter can be exchanged (it has Luer lock fittings at either end and is available as a spare part), but this obviously interrupts fluid resuscitation. The heat exchanger column can also become obstructed with clot with very large transfusions. The manufacturer now supplies inexpensive 340- $\mu$ m prefilters (adsorption type) which can be placed between the blood bag and the spike of the giving set. These dramatically reduce the quantity of debris reaching the main filter without slowing the rate of flow (personal communication. W. Verkaart, Level 1 Technologies).

Overall, we have found the Level 1 Fluid Warmer to be an extremely valuable piece of equipment and would recommend that one be available wherever major, rapid transfusions are likely to be required. This might include operating facilities that deal with emergency abdominal aortic aneurysms and severe trauma, as well as the more obvious but less common procedures, such as liver transplants.

MIEMSS Shock Trauma Center,  
Baltimore, MD, 21201,  
USA

J.P. NOLAN  
A.A.C. DOW

# The Datex Ultima - when you need the ultimate in safe anaesthesia care.



The new Datex Ultima is the flexible response to tightening monitoring standards.

It integrates all the parameters of gas exchange *and* ventilator function helping you to ensure complete patient safety during anaesthesia.

The new Ultima measures and displays CO<sub>2</sub>, O<sub>2</sub>, N<sub>2</sub>O and anaesthetic agents on the large, easy-to-read screen.

And since patient safety in routine anaesthesia is primarily dependant on adequate oxygenation, the fast paramagnetic Oxygen sensor in the Ultima measures inspired *and* expired O<sub>2</sub> data together with a breath-by-breath waveform display.

Additionally, with the new Ultima you can add pulse oximetry, agent identification, airway pressure and tidal volume – as and when you need them.

The Ultima also incorporates the innovative D-fend membrane water-trap eliminating the moisture problems which plague other airway gas monitors.

To find out how the new Datex Ultima can help you provide safe anaesthesia care ring your local S&W Vickers office on the number below.

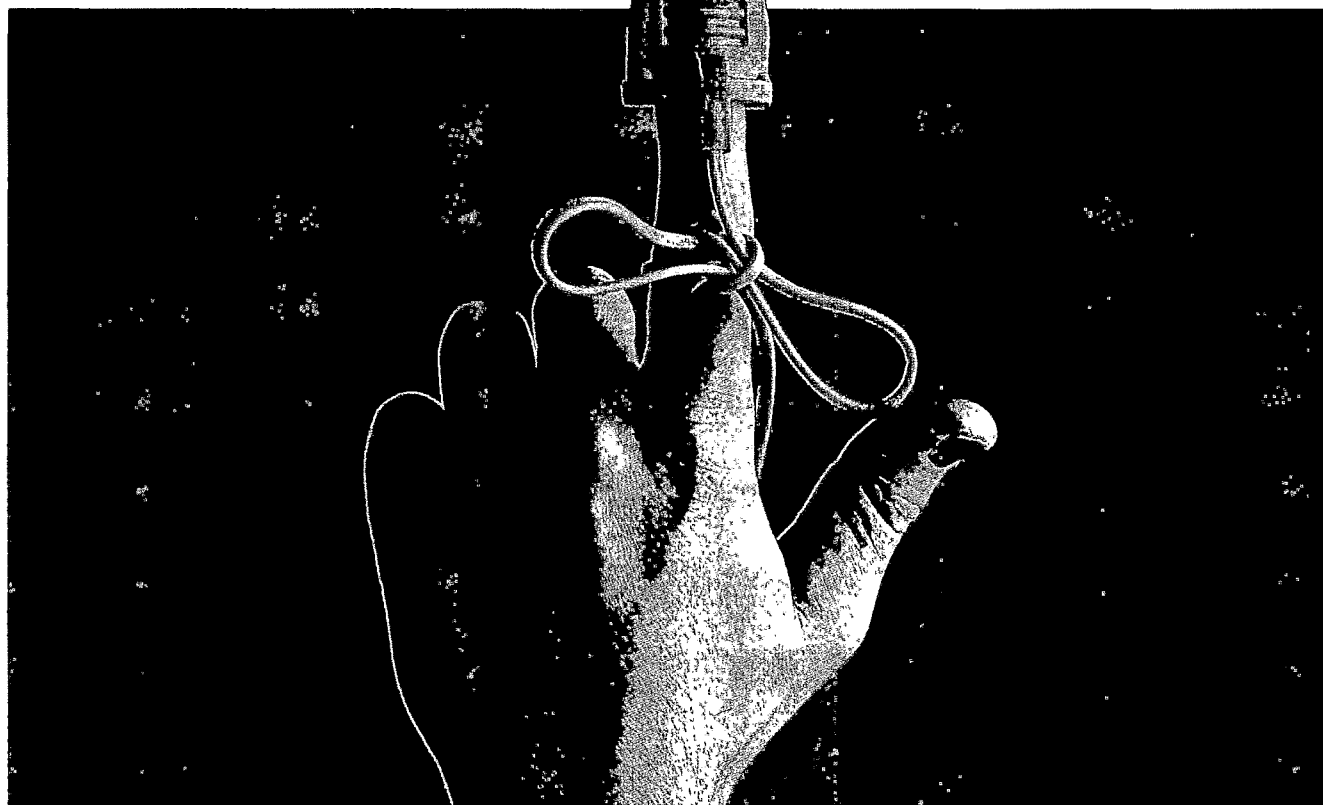
## S&W Vickers

Ruxley Corner, Sidcup, Kent, DA14 5BL. Tel 081-309 0433 Fax 081-309 0919

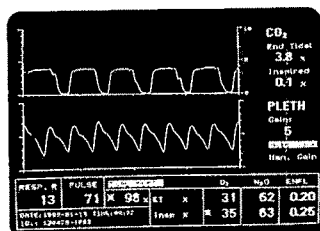
32 Kerse Road, Stirling, FK7 7TA. Tel 0786 51602 Fax 0786 51603

4 Balmoral Business Park, Boucher Crescent, Belfast. Tel 0232 669266

# If you're looking for a gas monitor, here's a feature worth remembering



**I**t's the pulse oximeter probe of Bruel & Kjaer's new Anesthetic Gas Monitor Type 1304, the multigas analyzer with a built-in pulse oximeter. The 1304 gives you a precise



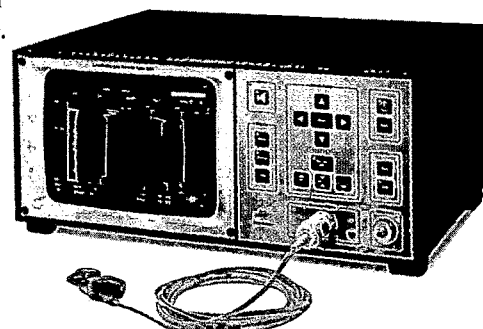
*Graphs of two functions can be displayed simultaneously.*

reading of peripheral arterial oxygen saturation (SpO<sub>2</sub>) and a stable plethysmographic curve — an invaluable aid for determining the quality of your SpO<sub>2</sub> reading and for trending changes in peripheral circulation. Besides SpO<sub>2</sub>, it measures O<sub>2</sub>, CO<sub>2</sub>, N<sub>2</sub>O, the inhalation anes-

thetic agents, pulse, and the rate of respiration.

All gas concentrations are measured simultaneously and continuously. They can be displayed as real-time waveforms with a true time relationship between the gases or as trend curves with a trend time of up to 16 hours. Which is worth remembering, too.

Call for full details.



**Brüel & Kjaer**   
**Brüel & Kjaer (UK) Ltd**

HEAD OFFICE: Harrow Weald Lodge · 92 Uxbridge Road · Harrow · Middlesex · HA3 6BZ  
Telephone: 081 954 2366 · Telex: 934150 · Fax: 081 954 9504

REGIONAL OFFICE: 86 East Road · Longsight · Manchester · M12 5GY · Telephone: 061 224 7590 · Fax: 061 248 6140

## References

1. SMITH JS, SNIDER MT. An improved technique for rapid infusion of warmed fluid using a Level 1 fluid warmer. *Surgery, Gynecology and Obstetrics* 1989; **168**: 273-4.
2. DUTKY PA, STEVENS SL, MAULL KI. Factors affecting rapid

fluid resuscitation with large-bore introducer catheters. *Journal of Trauma* 1989; **29**: 856-60.

3. LUNA, GK, MAIER RV, PAVLIN EG, ANARDI D, COPASS MK, ORESKOVICH MR. Incidence and effect of hypothermia in seriously injured patients. *Journal of Trauma* 1987; **27**: 1014-8.

## Blood or fluid warmers?

We read with interest the article by Drs Browne, De Boeck and Morgan evaluating the performance of the new Level 1 warmers (*Anaesthesia* 1990; **45**: 960-3). Their results are very similar to those obtained by us from our study of the Level 1 model 250.<sup>1</sup> Browne *et al.* report an outlet temperature above 34°C for the model 250 at a flow of 250 ml/minute which declined almost linearly as flow was increased above 250 ml/minute. By contrast, we measured a slightly lower outlet temperature of 33.6°C at a flow of 225 ml/minute which decreased sharply when the flow was increased above 250 ml/minute. Although the initial temperature of the infusate tested was similar in both studies, Dr Browne's group used saline while our group used packed red blood cells diluted with saline (haematocrit = 30%). This may explain in part the difference in our results, since heat transfer in a warming device is dependent on the density and specific heat capacity of the infusate.<sup>2</sup> This was demonstrated by Flancbaum *et al.* who evaluated the Level 1 model 500 and found that it was capable of warming 4°C saline to 32°C at a flow of 900 ml/minute.<sup>2</sup> However, when cold (4°C)

packed red blood cells were warmed to 32°C, the flow was limited to 440 ml/minute. Presumably, the performance when warming packed red blood cells that have been diluted with saline (which is the most likely case when the patient requires rapid transfusion) lies somewhere between these two values. When comparing the performances of warming devices, it is important to consider that the efficacy of these devices will depend in part on the type of infusate tested.

Riley Children's Hospital,  
Indianapolis, IN 46202-5200,  
USA

R.G. PRESSON, JR  
S.C. HILLIER  
K.A. HASELBY

## References

1. PRESSON RG JR, HASELBY KA, BEZRUCZKO AP, BARNETT E. Evaluation of a new high-efficiency blood warmer for children. *Anesthesiology* 1990; **73**: 173-6.
2. FLANCAUM L, TROOSKIN SZ, PEDERSEN H. Evaluation of blood-warming devices with the apparent thermal clearance. *Annals of Emergency Medicine* 1989; **18**: 355-9.

## Novel use of the Rendell-Baker Soucek mask

A patient, scheduled for radical neck dissection because of a recurrence of laryngeal carcinoma, had an end tracheostomy stoma from previous surgery and arrived in the anaesthetic room with no tracheostomy tube in place. Impressively smooth induction was aided by the use of a

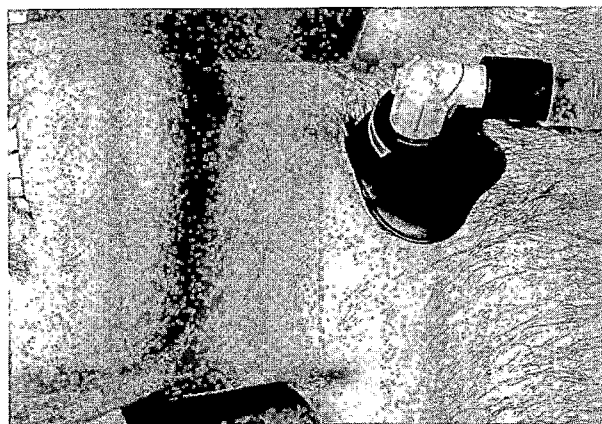


Fig. 1

size one Rendell-Baker Soucek mask, which was placed over the airway stoma, nasal projection inferiorly (Fig. 1). A good airtight seal was obtained and the mask was connected to a Bain system via a paediatric connector. In this manner pre-oxygenation was carried out comfortably and following intravenous injection of thiopentone and vecuronium a cuffed tracheostomy tube was inserted easily without any hypoxia or coughing. This was due to inflation of the lungs, achieved by squeezing the reservoir bag in the usual manner, until full relaxation was obtained.

Gas leaks via the pharynx in patients with a simple tracheostomy can be prevented by the opposition of the relaxed tongue to the posterior pharyngeal wall, and this technique is still valid. The senior author (M.J.W.) has also used the arrangement to deliver simple inhalation anaesthesia for induction and for minor surgical procedures in patients with a tracheostomy. We consider this technique a very useful addition to our anaesthetic practice.

34 Adel Green,  
Adel,  
Leeds LS16 8JX

D. NORTHWOOD  
M.J. WADE

## Anaesthesia in the field

Restall and colleagues investigated flumazenil administration after ketamine/midazolam anaesthesia (*Anaesthesia* 1990; **45**: 938-40). The placebo group recovery time varied from 5-72 minutes, which suggests

that a significant minority of patients would have become aware if the infusion had been stopped 10-15 minutes before the end of surgery, as recommended for the routine use of a very similar technique.<sup>1</sup> It would therefore seem

wise to continue the infusion to the completion of surgery, a practice which would extend the recovery time from the 12 minute mean originally reported<sup>1</sup> towards the 29 minute figure described by these authors and correctly considered by them to be unacceptable in the field. It is also apparent that recovery time is extremely unpredictable. For these and other reasons it is considered that the ketamine/midazolam/vecuronium technique, although novel and ingenious, is perhaps inappropriate for primary field anaesthesia.

To facilitate secondary surgery, Restall *et al.* report a technique employing ketamine, midazolam, isoflurane and spontaneous respiration (*Anaesthesia* 1990; 45: 965–8). The authors point out that field anaesthesia must be simple, using the minimum of equipment and yet report a technique which fails to achieve either of these objectives. Isoflurane administration from the Triservice apparatus (TSA) is theoretically suitable for both primary and secondary surgery,<sup>2</sup> so that there is little advantage to be gained by the addition of ketamine and midazolam, particularly when complicated by continuous infusion. If supplementation is considered necessary, the simplicity of intravenous morphine or neural blockade is to be preferred. The irritant nature of isoflurane can be easily overcome by a short period of manual ventilation.<sup>2</sup> The only real problem is cost, which is not of paramount importance in the overall military context. It is a pity that the proposed ketamine/midazolam/isoflurane technique was not compared with isoflurane alone or supplemented as described above, rather than to the redundant halothane/trichloroethylene combination.

The claim that ketamine is better than thiopentone for anaesthetic induction of shocked patients has never been substantiated in a controlled human study. In hypovolaemic pigs, ketamine and thiopentone both induced a similar decrease in blood pressure<sup>3</sup> and grossly unfavourable haemodynamic indices have been reported after ketamine anaesthesia of the critically ill.<sup>4</sup> Ketamine increases myocardial oxygen consumption more than supply<sup>5</sup> and induces greater lactic acidosis than thiopentone.<sup>3</sup> Therefore, ketamine administration could be more detrimental than a carefully titrated dose of thiopentone in hypovolaemia. Finally, the suggestion that the proposed technique is suitable for head injuries cannot be supported without more data. In the meantime, it is suggested that these patients are paralysed and hyperventilated in preference to any technique employing spontaneous ventilation and that in these circumstances isoflurane is very suitable alone.<sup>6</sup>

Royal Naval Hospital Haslar,  
Gosport,  
Hants

S.Q.M. TIGHE

## References

1. RESTALL J, TULLY AM, WARD PJ, KIDD AG. Total intravenous anaesthesia for military surgery. A technique using ketamine, midazolam and vecuronium. *Anaesthesia* 1988; 43: 46–9.
2. TIGHE SQM, PETHYBRIDGE RJ. A comparison of halothane and trichloroethylene with isoflurane. A study of drawover air anaesthesia with the Triservice anaesthetic apparatus. *Anaesthesia* 1987; 42: 887–91.
3. WEISKOPF RB, BOGETZ MS, ROIZEN MF, REID IA. Cardiovascular and metabolic sequelae of inducing anaesthesia with ketamine or thiopental in hypovolemic swine. *Anesthesiology* 1984; 60: 214–9.
4. WAXMAN K, SHOEMAKER WC, LIPPMAN M. Cardiovascular effects of anesthetic induction with ketamine. *Anesthesia and Analgesia, Current Researches* 1980; 59: 355–8.
5. FOLTS, JD, AFONSO S, ROWE GG. Systemic and coronary haemodynamic effects of ketamine in intact anaesthetised and un-anaesthetised dogs. *British Journal of Anaesthesia* 1975; 47: 686–94.
6. FROST EAM. Inhalation anaesthetic agents in neurosurgery. *British Journal of Anaesthesia* 1984; 56: 47–55S.

## A reply

Thank you for the opportunity to reply to Dr Tighe's letter on the use of ketamine/midazolam infusions for field anaesthesia.

Dr Tighe suggests that a significant minority of patients would have been aware if the infusion had been stopped 10–15 minutes before the end of surgery. We would submit that, with our method as with most other anaesthetic techniques, recovery time is related to the duration of anaesthesia. In one of our previous studies<sup>1</sup> a mean recovery time of 12 minutes occurred after a mean induction of anaesthesia to end of operation time of approximately 20 minutes and at that time we recommended that the muscle relaxant component was infused separately to allow easy reversal without lightening of anaesthesia. It is our contention that the infusion should be continued to the end of short cases i.e. those lasting less than 30 minutes. In cases lasting longer than 30 minutes, in view of our published data,<sup>1,2</sup> it is not unreasonable to discontinue the infusion 10–15 minutes before the end without undue risk of awareness and with a consequent shortening of recovery time.

The object of our paper on the use of ketamine/midazolam in combination with isoflurane was to try and establish a technique which could be used for secondary surgery without recourse to tracheal intubation. We compared our method with the well-tried and documented method of halothane and trichloroethylene in the Tri-Service Anaesthetic Apparatus (TSA).<sup>3,4</sup> Other workers, including Dr Tighe, have found no difference in recovery times between halothane/trichloroethylene and isoflurane using the TSA.<sup>5,6</sup> The use of isoflurane/trichloroethylene with spontaneously breathing patients using the TSA was found to produce inadequate anaesthesia in eight out of 20 patients and additional supplementation was required.<sup>6</sup>

We have never claimed that ketamine is superior to thiopentone in the induction of trauma patients. However, it has been used widely in the induction of injured patients including many who were wounded in the Falklands conflict.<sup>7</sup> In the first study quoted by Dr Tighe,<sup>8</sup> hypovolaemic pigs maintained  $\text{DO}_2$  and  $\text{VO}_2$  with ketamine and thiopentone. The lactate levels in both groups were not significantly different 90 minutes after transfusion, which supports our contention that the choice of induction agent is no substitute for vigorous and adequate pre-operative resuscitation. The second study is not comparable.<sup>9</sup> The patients studied by Waxman *et al.*<sup>9</sup> were elderly, suffering from septic shock and *in extremis*. We are advocating the use of ketamine in the previously fit, resuscitated, injured soldier, who has a reasonable chance of survival.

Dr Tighe claims that isoflurane delivered from the TSA is theoretically suitable for both primary and secondary field surgery. We believe that a technique which employs thiopentone induction followed by ventilation with a relatively high concentration of isoflurane for 3 minutes followed by further ventilation with a lower concentration for up to 6 minutes in a possibly hypovolaemic patient will not produce cardiovascular stability. Indeed, his results show a significantly lower arterial blood pressure at 13–18 minutes postinduction after surgical stimulation. We would have been interested in the cardiovascular changes in the immediate postinduction period.



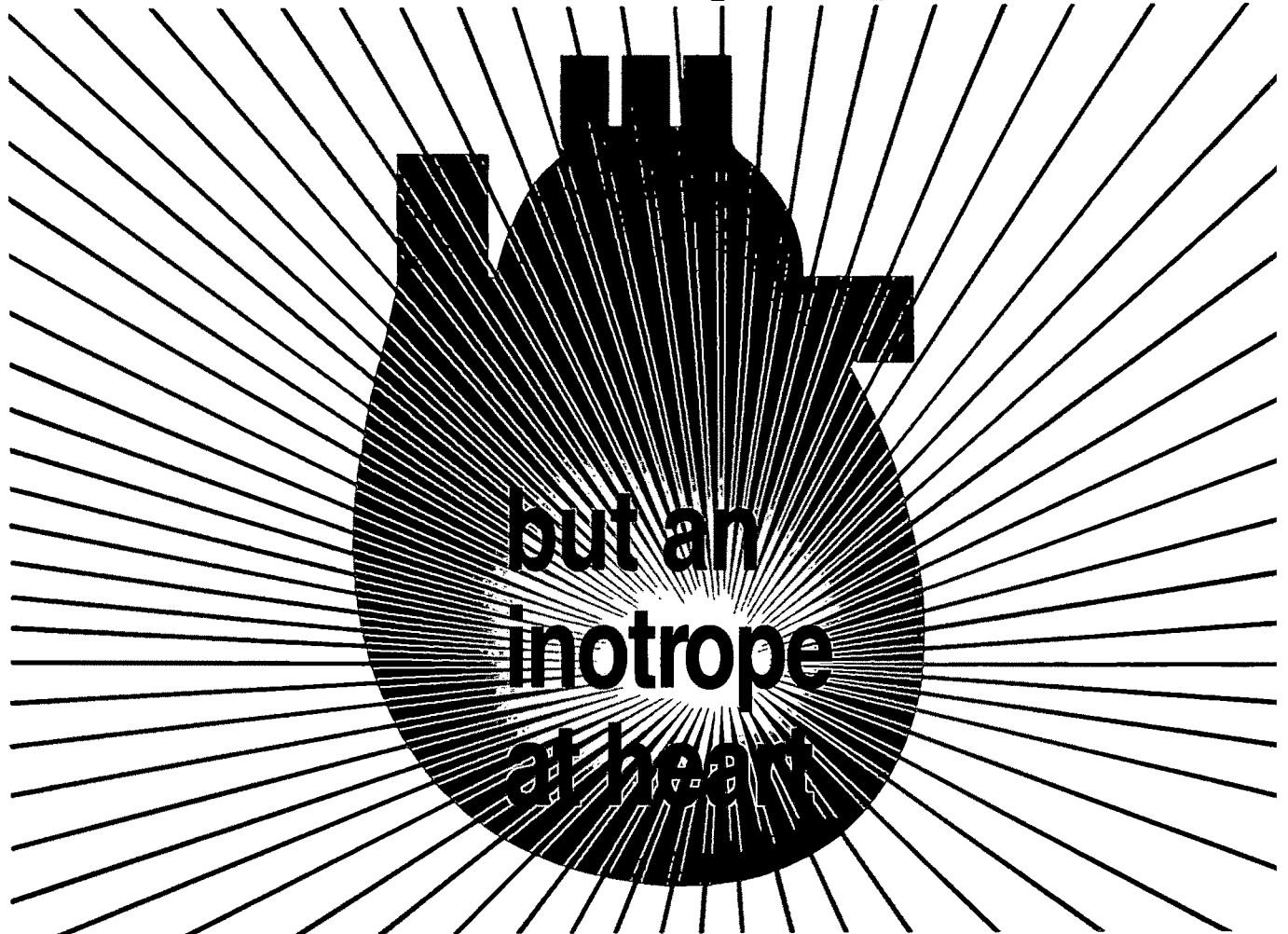
*Now - more strength meets less resistance*



# **PRIMACOR**

**milrinone**

**a vasodilator peripherally...**



**balanced intravenous support  
in resistant chronic heart failure**



**strengthens systolic  
function<sup>1-3</sup>**



**reduces systemic<sup>4-6</sup> and  
pulmonary<sup>7</sup> vascular  
resistance**



**increases venous  
capacitance<sup>4</sup>**



**increases output at reduced fill  
pressures<sup>8</sup>**



**with myocardial oxygen supply  
matching demand<sup>9</sup>**

**Sterling Winthrop**

Prescribing information and  
references on adjacent page.

# **PRIMACOR**

**strengthens contraction, reduces resistance**



**milrinone**

## References

1. *Circulation* 1986; **73** (1): 130-137
2. *Circulation* 1986; **73** (3/2 Suppl): 175-183
3. *J Am Coll Cardiol* 1987; **10** (6): 1294-1302
4. *J Am Coll Cardiol* 1986; **8** (2): 413-418
5. *J Am Coll Cardiol* 1987; **9** (4): 711-722
6. *Am J Cardiol* 1987; **59** (15): 1345-1350
7. *Am J Cardiol* 1987; **60** (16): 1329-1333
8. *N Eng J Med* 1983; **309** (13): 748-756
9. *Circulation* 1985; **71** (5): 972-979

## Prescribing information ▼

For the short term treatment of severe congestive heart failure unresponsive to conventional maintenance therapy.

Ampoules containing 10mg milrinone as the lactate in 10ml solution for intravenous use.

**Basic NHS cost:** Carton of 10 ampoules £147.44.

PL 0071/0323

**Dosage in adults including elderly:** Loading with 50 micrograms/kg intravenously over 10 minutes then infusion within the range 0.375 to 0.75 micrograms/kg per minute according to clinical and haemodynamic response to a daily maximum of 1.13mg/kg. In impaired renal function use lower infusion rate (see Data Sheet or Package Insert Leaflet).

**Contraindications:** Hypersensitivity to milrinone.

**Precautions:** Not recommended immediately following acute myocardial infarction, in place of surgical relief of outlet obstruction nor in the presence of uncontrolled atrial flutter or fibrillation. Close clinical, haemodynamic, electrophysiological, fluid, electrolyte and renal function monitoring is advised in all patients. Slow or stop infusion where excessive decrease in blood pressure occurs; correct hypokalaemia by prophylactic potassium supplementation. Use with caution where filling pressures are reduced e.g. by prior vigorous diuretic therapy. In pregnancy and lactation use only if potential benefit justifies potential risk to fetus or infant.

**Side Effects:** Hypotension, angina and headaches may occur. Supraventricular and ventricular arrhythmias, hypokalaemia, tremor and reduction in platelet count have been reported.

**Drug Interactions:** There is a theoretical potential interaction with calcium channel blockers.

**Chemical compatibility:** Do not administer in same intravenous line as other drugs; frusemide and bumetanide may precipitate if mixed with milrinone infusion.

Primacor is a registered trade mark. For full prescribing information see Data Sheet.

Further information available from:

Sterling-Winthrop Group Ltd  
Onslow Street, Guildford,  
Surrey GU1 4YS

Our technique was originally described for use in operating theatres which had limited ventilation in a nuclear, biological or chemical warfare threat. We would be interested to know how Dr Tighe would use isoflurane from an uncompensated vaporizer in temperatures of 45°C–53°C which have been recorded in our operating theatres in the Gulf. Isoflurane boils at 48.5°C. We do not propose that ketamine, midazolam and isoflurane should be used for spontaneously breathing patients undergoing neurosurgery. We cannot emphasize enough that modern battle casualties are expected to suffer multiple injuries. This technique is suitable for patients who may have, or have had, concomitant head injuries.

Finally, we do not believe that the use of a syringe pump and infusion is unnecessarily complicated for use in the field. We have now instructed a large number of combat anaesthetic support officers (CASOs) in its use and they all find it easy and simple to use.

Cambridge Military Hospital,  
Aldershot GU11 2AN

J. RESTALL  
I.G. JOHNSTON

#### References

1. BAILIE R, CRAIG G, RESTALL J. Total intravenous anaesthesia for laparoscopy. *Anaesthesia* 1989; **44**: 60–3.

2. RESTALL J, TULLY AM, WARD PJ, KIDD AG. Total intravenous anaesthesia for military surgery. A technique using ketamine, midazolam and vecuronium. *Anaesthesia* 1988; **43**: 46–9.
3. KNIGHT RJ, HOUGHTON IT. Field experience with the Tri-Service apparatus in Oman and NI. *Anaesthesia* 1981; **36**: 1122–7.
4. BULL PT, MERRILL SB, MOODY RA, BAKER DJ, YATES A, GERAGHTY IF, TIGHE SQM, SQUIRES SJ, STOTT CJ. Anaesthesia during the Falklands campaign. The experience of the Royal Navy. *Anaesthesia* 1983; **38**: 770–5.
5. TIGHE SQM, PETHYBRIDGE RJ. A comparison of halothane and trichlorethylene with isoflurane. A study of drawover air anaesthesia with the Tri-Service anaesthetic apparatus. *Anaesthesia* 1987; **42**: 887–91.
6. KOCAN M. The Tri-Service anaesthetic apparatus. *Anaesthesia* 1987; **42**: 1101–4.
7. JOWITT MD, KNIGHT RJ. Anaesthesia during the Falklands campaign. The land battles. *Anaesthesia* 1983; **38**: 776–83.
8. WEISKOPF RB, BOGETZ MS, ROIZEN MF, REID IA. Cardiovascular and metabolic sequelae in inducing anaesthesia with ketamine or thiopental in hypovolaemic service. *Anesthesiology* 1984; **60**: 214–9.
9. WAXMAN K, SHOEMAKER WC, LIPPMAN M. Cardiovascular effects of anaesthetic induction with ketamine. *Anesthesia and Analgesia* 1980; **59**: 355–8.

### The laryngeal mask airway in paediatric anaesthesia

Johnston *et al.* report the use of the Brain laryngeal mask airway in paediatric otological anaesthesia (*Anaesthesia* 1990; **45**: 924–7). Although this study demonstrates the value of the laryngeal mask in this setting it also highlights some problems. The authors clearly demonstrate by their figures for hypoxic episodes that the airway was not well maintained by the use of a facemask during these brief (<12.5 minutes) anaesthetics. Surely the ability to maintain an adequate airway by mask is one of the fundamental skills of anaesthetic practice?

The authors would appear to have made life difficult for themselves by using Guedel airways in only four children in the facemask group. Discussion with other senior registrars in our department who are familiar with paediatric anaesthesia, revealed that we all insert a Guedel airway automatically when performing a mask general anaesthetic in a child who will be under surgical drapes and will have the head turned during the anaesthetic. Head-turning with a laryngeal mask or a tracheal tube in place will surely increase the risk of sore throat. Johnston *et al.* report that four children (16.6%) complained of sore throat after use of the laryngeal mask. This figure may be an underestimate since many of their group were too young to respond, but it is still higher than that previously reported for the laryngeal mask (7–12%)<sup>1,2</sup> or facemask.<sup>2</sup>

In a recent article Mason and Bingham<sup>3</sup> reported the value of the laryngeal mask, while emphasising the importance of antisialogogue premedication (not given in the current day case study) and of secure fixation. The laryngeal mask airway is certainly useful in freeing the anaesthetist's hands but at the same time it distances him or her from the patient. This is particularly inadvisable when the surgeon needs the head turned to reach the second ear, at which point a laryngeal mask, albeit apparently well fixed, may be disturbed under the drapes and lead to obstruction. Holding a facemask allows the

anaesthetist to be in direct contact with the patient with a finger on the pulse, a monitoring tool which is so often forgotten in modern hi-tech anaesthetic practice.

St. Thomas' Hospital,  
London SE1 7EH

D.E. WITHINGTON

#### References

1. BRODRICK PM, WEBSTER NR, NUNN JF. The laryngeal mask airway. A study of 100 patients during spontaneous breathing. *Anaesthesia* 1989; **44**: 238–41.
2. ALEXANDER CA, LEACH AB. Incidence of sore throats with the laryngeal mask. *Anaesthesia* 1989; **44**: 791.
3. MASON DG, BINGHAM RM. The laryngeal mask airway in children. *Anaesthesia* 1990; **45**: 769–3.

#### A reply

Thank you for asking us to reply to the letter from Dr Withington. We are pleased that he accepts the overall conclusion concerning the use of the laryngeal mask airway (LMA) in paediatric otological surgery. The observation concerning the insertion of an oral airway must be interpreted in the light of the fact that all the anaesthetics in this study were given by anaesthetists experienced in this type of paediatric surgery; we do not believe that in all such cases there is a need for a Guedel airway to be used routinely. Whether head-turning with a laryngeal mask or tracheal tube *in situ* increases the risk of a sore throat is supposition. The incidence of sore throat, in our study, in both groups of children was of the same order of magnitude, four (LMA) versus two (facemask), in keeping

with the similar results of Alexander and Leach.<sup>1</sup> We would agree that, in comparison with a facemask or LMA, tracheal intubation in adults<sup>2</sup> and children<sup>3</sup> leads to an increased risk of laryngeal oedema and trauma. Our recent work demonstrates the increased risk of trauma with tracheal intubation in comparison with the LMA for airway maintenance.<sup>2</sup>

We would agree with Dr Withington that the anaesthetist is responsible for supervising all the movements of an anaesthetised patient and particularly for any re-arrangement of the airway, but in keeping with other workers,<sup>4</sup> we consider the freeing of the anaesthetists hands during surgery to be advantageous.

Whilst there are many ways of managing the same problem, this study demonstrates the difficulty of the maintenance of an airway with a facemask during this type of surgery, due to the close proximity of the surgical field to the airway and the need for re-arrangements of the airway during the procedure. We would reiterate that in this type of surgery the LMA has advantages for airway

maintenance compared with a facemask for patient safety and surgical operating conditions.

*Guy's Hospital,  
London SE1 9RT*

S.F. WRIGLEY  
H.E. JONES  
D.F. JOHNSTON  
P.F. ROBB

#### References

1. ALEXANDER CA, LEACH AB. Incidence of sore throats with the laryngeal mask. *Anaesthesia* 1989; **44**: 791.
2. HARRIS TM, JOHNSTON DF, COLLINS SRC, HEATH ML. A new general anaesthetic technique for use in singers: The Brain layngeal mask airway versus endotracheal intubation. *Journal of Voice* 1990; **4**: 81-5.
3. GLEESON, MJ, FOURCIN, AJ. Clinical analysis of laryngeal trauma secondary to intubation. *Journal of the Royal Society of Medicine* 1983; **76**: 928-32.
4. BRODRICK PM, WEBSTER NR, NUNN JF. The laryngeal mask airway. A study of 100 patients during spontaneous breathing. *Anaesthesia* 1989; **44**: 238-41.

#### Exploding ampoules

I would like to bring readers' attention to a potential hazard in the preparation of intravenous injections in rubber-sealed glass ampoules.

I was in the operating theatre preparing some flucloxacillin (Floxapen 500 mg, Beecham) by the addition of sterile water. There was a loud crack after injection of a small amount of water and the ampoule exploded. A search revealed the remains of the ampoule some distance away on the floor and showed that the base of the ampoule had been neatly separated from the rest; only 3 ml of water had been injected. Examination of the base showed that the ampoule was thinned around the whole of its perimeter, obviously due to a manufacturing fault.

Subsequent measurements on similar Floxapen ampoules have revealed that their capacity is approximately 7.5 ml and their contents are at 18-20 mmHg above atmospheric pressure, i.e. 780 mmHg absolute. Injection of 3 ml water would be expected to increase the internal pressure by approximately 520 mmHg. (Boyle's Law;  $p_1 \cdot V_1 = p_2 \cdot V_2$ ,  $P_2 = (780 \times 7.5)/4.5$ ,  $P_2 = 1300$  mmHg). A well-produced ampoule can withstand this stress easily but a badly manufactured vessel obviously does not. The risk of fracture will be highest with small ampoules where relatively high internal pressures are produced due to their small internal volume and because they are often made from thinner glass.

Many readers are, no doubt, already well aware of the problem usually associated with the enthusiastic injection of too large a volume of liquid into a closed ampoule in an attempt to achieve a rapid solution of a drug, i.e. an abrupt hissing sound is accompanied by topical application of the drug over personnel in the immediate vicinity, as needle becomes separated from syringe. On this occasion, however, the volume of liquid injected was not excessive, but the results were just as spectacular and considerably more dangerous. The immediate hazards of this event were loss of glass fragments into the patient's operation site and risk to medical personnel from flying splinters of glass. Fortunately, neither occurred. Since it would seem likely that the occasional defective ampoule will continue to be produced and as there is no simple method for detecting them, it would be as well to take whatever precautions we can to protect ourselves and the patient.

A few common-sense measures could ensure prevention of this hazard. I would recommend that drugs prepared by injecting fluid into rubber-sealed glass ampoules are made up in the anaesthetic room rather than the operating theatre, to exclude the risk of fragments entering the surgical site should an ampoule disintegrate. Frequent aspiration of small volumes of air from within the ampoule similar to the volume of fluid being injected, should prevent a significant rise in the pressure within the ampoule and finally the drug should be prepared while facing away from other medical personnel and, perhaps most importantly, away from the preparer's eyes to avoid injury in the event of an ampoule shattering.

*Queens Medical Centre,  
Nottingham NG5 2UA*

S. WILLIAMS

#### A reply

This is a regrettable but rare occurrence. Every year 20 million ampoules leave our antibiotic manufacturing site and in the last 10 years we have received only one similar complaint. This case involved an Ampiclox vial, the base of which broke off from the body of the vial, although not in the explosive manner described. Examination of the pieces of 'Ampiclox' vial revealed a visible impact point consistent with the vial receiving a blow of some kind.

We regularly sample the vials we use, to check dimensions and test for strain; however, with such a low defect rate it is unlikely that every defective vial will be identified in this way. Our many years of experience have shown that it is unusual for weak vials to survive the washing, capping and packing processes or, indeed, the final transit stage. It is conceivable, however, that some weak vials may remain intact up to the time of use.

Unfortunately we are unable to make any specific comment on this particular case without a detailed examination of the vial fragments.

*UK Product Marketing,  
Beecham Research,  
Welwyn Garden City AL7 1EY*

A.M. COTTERELL



#### Prescribing information

**Indications and dosage:** Systemic candidiasis: 400mg on the first day followed by 200-400mg once daily. Cryptococcosis, including meningitis: 400mg on the first day followed by 200-400mg once daily. Maintenance therapy to prevent relapse of cryptococcal meningitis in patients with AIDS: at least 100mg daily. Oropharyngeal candidiasis: 50-100mg once daily for 7-14 days or longer in immunocompromised patients. Other mucosal candidal infections: 50-100mg once daily for 14-30 days. Vaginal candidiasis: single 150mg dose. Use in the elderly: as above except for those renally impaired - see data sheet. Use in children - not recommended. **Administration:** Diflucan may be administered either orally or by intravenous infusion at a rate of approximately 5-10ml/min. The dosages for the two routes are equivalent. **Contra-indications:** Hypersensitivity to fluconazole or related triazoles, pregnancy and women of childbearing potential unless adequate contraception is employed.

**Warnings:** Lactation: Not recommended. Renal impairment: dosage reduction may be necessary, see data sheet. **Drug interactions:** Monitor patients on concurrent anticoagulants, oral sulphonylureas, phenytoin or rifampicin. **Side-effects:** Nausea, abdominal discomfort, diarrhoea and flatulence. **Package Quantities and basic NHS Cost:** 50mg capsule, calendar pack of 7, £16.61 (PL 57/0289); 200mg capsule, calendar pack of 7, £66.42 (PL 57/0317); 150mg capsule, pack of 1, £7.12 (PL 57/0290); Bottles of 25ml and 100ml containing Diflucan 2mg/ml intravenous infusion - 25ml (50mg) bottle, £7.32; 100ml (200mg) bottle, £29.28 (PL 57/0315). Hospital prices are available on request. **References:** 1 - 4 Data on file, Pfizer Ltd. Further information on request.



Pfizer Limited Sandwich, Kent

# Antifungal therapy that's severe on the infection, not on the patient.

Diflucan Oral/I.V. is a highly effective, well tolerated therapy with a convenient, once a day dosage and administration.

Overall clinical responses in deep-seated fungal infections are impressive: 87% in systemic candidiasis<sup>1</sup>, 96% in mucosal candidiasis<sup>2</sup> and 62% in cryptococcal meningitis.<sup>3</sup> Tolerability is similarly impressive with adverse effects comparable to placebo.<sup>4</sup>

Diflucan Oral/I.V. Treating invasive fungal infection with an iron fist and patients with a velvet glove.

**Diflucan** ORAL/I.V.  
fluconazole \*Trade Mark  
Counters invasive fungal infection



## JMD/0390/A788



## Disposable carbon dioxide detectors

Considerable enthusiasm has been voiced recently for the use of a disposable carbon dioxide detector (FEF<sup>TM</sup> End-Tidal CO<sub>2</sub> Detector, Fenem Inc) to detect inadvertent oesophageal intubation during anaesthesia (*Anaesthesia* 1990; **45**: 465-7 and 653-5) and for attempted resuscitation.<sup>1,2</sup> We believe that a degree of reality should be injected into the discussions regarding this item. During anaesthesia, unrecognised oesophageal intubation remains a problem with potentially catastrophic consequences. It is alleged that even visual confirmation of correct tube placement, the presence of normal chest movements and breath sounds and the absence of breath sounds during epigastric auscultation may be misleading signs of tracheal intubation under certain circumstances.<sup>3</sup> However, provided that cardiac output, pulmonary circulation and carbon dioxide production are constant, the presence of carbon dioxide in expired respiratory gas predicts accurately tracheal intubation. It is, of course, the undetected, rather than the immediately obvious oesophageal intubation that most risks morbidity and mortality. Therefore, if one accepts that all 'simple' methods are fallible, any method of detecting such an occurrence, if it is to have any influence on the incidence of undetected oesophageal intubation, must be employed on every occasion and not merely when the anaesthetist is suspicious of the tube position. In the two hospitals in our health district there were approximately 12 000 intubations in the operating theatres in 1989. Consequently, to use one FEF device for each intubation, at an individual price of approximately £13.00, would entail an annual expenditure of approximately £156 000. This money would be better spent on purchasing capnometers, approximately 50 of which could be bought with the same amount of money; each capnometer would also have a life-span of approximately 10 years.

The recent suggestion by O'Flaherty and Adams commending the use of the FEF device that, in the UK, the use of dedicated anaesthetic rooms 'separates the anaesthetist from capnography during induction when it is most informative' does little to promote the desired improvement in anaesthetic room monitoring standards.<sup>4</sup> Monitoring equipment, including a capnometer, on a trolley is easily moved between the anaesthetic room and operating theatre. For these reasons the requirement for a disposable carbon dioxide detector for use during anaesthesia does not seem to be either cost-effective or, indeed, entirely necessary.

Wilkinson *et al.* state<sup>5</sup> that 'The other area where this detector is of unique value is in cardiopulmonary resuscitation . . .'. However, we have demonstrated,<sup>6</sup> and Hayes *et al.* have confirmed,<sup>7</sup> that at a cardiac arrest the FEF end-tidal carbon dioxide is unreliable if, as still occurs in some institutions, a breathing system which allows rebreathing is used. In such circumstances the breath-to-breath colour change is lost and the indicator remains permanently yellow.<sup>6,7</sup> Also, the tracheal administration of atropine, adrenaline or lignocaine, as recommended by the UK Resuscitation Council<sup>8</sup> and NHS Training Authority manual on extended training in ambulance aid,<sup>9</sup> consistently leads to a permanent yellow change in the indicator paper of the FEF device<sup>6</sup>. In fact, the FEF device is not specific for carbon dioxide, being composed of a pH-sensitive indicator paper which consistently reacts with acid solutions *in vitro*; all drugs tested by us had a pH of less than 5.93. Further, acidic gastric aspirate also turns the indicator permanently yellow<sup>6</sup> or orange.<sup>7</sup>

Detection of carbon dioxide by the FEF detector also presupposes that there is an adequate cardiac output.

Indeed, the item has been recommended as a guide to the efficacy of cardiopulmonary resuscitation.<sup>1</sup> If cardiac output is absent or low, the indicator paper shows a continuous purple colour; this does not differentiate between a misplaced tracheal tube and reduced cardiac output or pulmonary perfusion. Ironically, it is then necessary to check correct placement of the tracheal tube; the only methods available are those 'simple' techniques whose efficacy is already in question. Again, we have considered the financial aspects involved in the regular use of this device at resuscitation attempts. In 1989, there were 540 cardiac arrest calls requiring intubation in the Portsmouth and SE Hants Health Authority. There are 113 cardiac arrest boxes in the two acute hospitals, entailing an initial outlay of approximately £1500 to place one detector in each box. There would be an annual replacement cost of £7020 for those used. Additionally, with a shelf-life of only 15 months, some detectors in 'low risk' areas would require renewal without use.

Tracheal intubation at cardiac arrests, although often desirable, is not essential. Ventilation is not synonymous with intubation and can be easily achieved using a bag-mask-airway technique, or mouth-to-mask technique via a Laerdal pocket mask enriched with oxygen. If the incidence of incorrect placement of tracheal tubes at cardiac arrests is as high as has been reported,<sup>3</sup> then it is probable that we should reconsider the training programmes offered in advanced airway support.

Unlike the situation in anaesthesia where capnometers are available for carbon dioxide detection, the FEF device may yet have a limited role at cardiac arrests. However, those using this device at such events should remember that the FEF end-tidal CO<sub>2</sub> detector does not monitor carbon dioxide specifically, and is not, as has been repeatedly suggested, either reliable or inexpensive.

Queen Alexandra Hospital,  
Portsmouth PO6 3LY

J.D. MUIR  
P.B. RANDALLS  
G.B. SMITH  
B.L. TAYLOR

## References

- HIGGINS D, HAYES M, DENMAN W, WILKINSON DJ. Effectiveness of using end tidal carbon dioxide concentration to monitor cardiopulmonary resuscitation. *British Medical Journal* 1990; **300**: 581.
- RODGERS RC. The FEF carbon dioxide analyser. *Anaesthesia* 1990; **45**: 591.
- BIRMINGHAM PK, CHENEY FW, WARD RJ. Esophageal intubation: a review of detection techniques. *Anesthesia and Analgesia* 1986; **65**: 886-91.
- ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND. Recommendations for Standards of Monitoring during Anaesthesia and Recovery. July 1988.
- WILKINSON DJ, HAYES M, DENMAN WP, HIGGINS D. The Fenem FEF CO<sub>2</sub> detector. *Anaesthesia* 1990; **45**: 694.
- MUIR JD, RANDALLS PB, SMITH GB. End-tidal carbon dioxide detector for monitoring cardiopulmonary resuscitation. *British Medical Journal* 1990; **301**: 41-2.
- HAYES M, YAU EHS, HIGGINS D, DENMAN W, WILKINSON DJ. End-tidal carbon dioxide detector for monitoring cardiopulmonary resuscitation. *British Medical Journal* 1990; **301**: 42.
- CHAMBERLAIN DA. Advanced life support. Revised recommendations of the Resuscitation Council (UK). *British Medical Journal* 1989; **299**: 446-448.
- AMBULANCE STAFF TRAINING COMMITTEE. Extended training in ambulance aid. Bristol NHSTA, 1987.

*A reply*

We consider that Dr Muir and colleagues have rather missed the points we tried to make regarding the Fenem CO<sub>2</sub> detector.

We clearly stated that 'we believe the capnograph, when available . . . remains the apparatus of choice to confirm accurate intubation. Until all sites have these devices, the Fenem CO<sub>2</sub> detector will provide vital information to personnel who attempt tracheal intubation'.<sup>1</sup> Other authors have also concluded that the detector should be recommended where capnography is not available.<sup>2</sup>

The most reliable methods for ensuring correct placement of a tracheal tube are either direct observation of passage of the tube into the trachea or the measurement of carbon dioxide in the expired gas mixture.<sup>3</sup> If the anaesthetist sees the tracheal tube pass through the vocal cords then, unless the patient is moved or repositioned, when the tube may become dislodged, there would be little point in attaching the detector to confirm placement. It is in those cases where visual confirmation is not possible and where a capnograph is not available that the device is useful. A report of one such occasion has been published.<sup>4</sup> Thus, in contrast to the view expressed above, we believe a role does exist for a disposable CO<sub>2</sub> detector in current anaesthetic practice.

With regard to use of the device during cardiopulmonary resuscitation, we found the identification of rebreathing to be of clinical value. Indeed, the first such colorimeter for measuring exhaled CO<sub>2</sub> in an anaesthetic system was designed specifically to detect such an occurrence by W.B. Draper in 1936.<sup>1</sup>

In the cardiac arrest situation, the Fenem CO<sub>2</sub> detector plays a valuable role in assessing the initial placement of the tracheal tube. Under such circumstances, any colour change will confirm correct tracheal intubation.<sup>5</sup> Adequate cardiac massage should produce end-tidal CO<sub>2</sub> levels of at least 0.5%,<sup>6</sup> which is sufficient to produce a detectable colour change.<sup>7</sup> If the detector does not change colour, then either the tracheal tube is in the oesophagus or the cardiac output is very low. In such an event, clinical judgement must prevail. If tracheal administration of atropine, adrenaline and lignocaine is to be relied upon during resuscitation, this initial confirmation of tracheal intubation will ensure that these drugs are not deposited into the oesophagus. During subsequent resuscitation, a cyclical colour change from purple to yellow indicates effective ventilation and cardiac massage. A fixed yellow colour indicates contamination of the device by drugs or acid aspiration. This is easily recognised.<sup>8,9</sup>

There are financial aspects to be considered before these detectors are placed in every crash box, and perhaps higher risk areas such as coronary care and the accident and emergency department could be given preferential treatment. Hospitals and budget holders must make up their own minds. We have never stated that intubation is the only method of ensuring ventilation at a resuscitation. Bag to mask or expired air ventilation is obviously still acceptable, but in skilled hands, the tracheal tube provides the added benefit of a secured airway with the lungs protected from aspiration.

We continue to find the Fenem CO<sub>2</sub> detector, when correctly used, a reliable and valuable addition to the anaesthetist's and resuscitator's armamentarium.

*St. Bartholomew's Hospital,  
London EC1A 7BE*

D.J. WILKINSON  
W.T. DENMAN  
D. HIGGINS  
M. HAYES  
E. YAU

*References*

1. DENMAN WT, HAYES M, HIGGINS D, WILKINSON DJ. The Fenem CO<sub>2</sub> detector device. *Anaesthesia* 1990; **45**: 465-7.
2. GOLDBERG JS, RAWLE PR, ZEHNDER JL, SLADEN RN. Colorimetric end-tidal carbon dioxide monitoring for tracheal intubation. *Anesthesia and Analgesia* 1990; **70**: 191-4.
3. STRUNIN L, WILLIAMS T. The FEF end-tidal carbon dioxide detector. *Anesthesiology* 1989; **71**: 621-2.
4. HIGGINS D, LLOYD-THOMAS AR. Confirmation of tracheal intubation in a neonate using the Fenem CO<sub>2</sub> detector. *Anaesthesia* 1990; **45**: 591-2.
5. GERARD J, MACLEOD BA, HELLER MB, YEALY DM. Verification of endotracheal intubation using a disposable end-tidal CO<sub>2</sub> detector. *Prehospital and Disaster Medicine* 1989; **4**: 74.
6. GARNETT AR, ORNATO JP, GONZALEZ ER, JOHNSON EB. End-tidal carbon dioxide monitoring during cardiopulmonary resuscitation. *JAMA* 1987; **257**: 512-5.
7. JONES BR, DORSEY MJ. Disposable end-tidal CO<sub>2</sub> detector: minimal CO<sub>2</sub> requirements. *Anesthesiology* 1989; **71**: A359.
8. MUIR JD, RANDALLS PB, SMITH GB. End-tidal carbon dioxide detector for monitoring cardiopulmonary resuscitation. *British Medical Journal* 1990; **301**: 41-2.
9. HAYES M, YAU EHS, HIGGINS D, DENMAN WT, WILKINSON DJ. End-tidal carbon dioxide detector for monitoring cardiopulmonary resuscitation. *British Medical Journal* 1990; **301**: 42.

*A reply*

Thank you for the opportunity to reply. We are grateful for these comments on our study. Most anaesthetists agree that 'simple' clinical methods of detection of tracheal intubation are fallible and that continuous display of the capnographic waveform is essential for the rapid diagnosis of the potentially catastrophic oesophageal tracheal tube misplacement. Until Health Authorities have the foresight to provide the funding for capnometry at all locations where anaesthesia is administered, the Fenem end-tidal carbon dioxide detector may have a role in reducing the risk of inadvertent oesophageal intubation. While accepting the potential cost savings, it is the anaesthetist's role to continue to emphasise to management, the potential financial, and more importantly, safety benefits of capnometry.

*Guy's Hospital,  
London*

D. O'FLAHERTY  
A.P. ADAMS

**Hypotension and brain death?**

It is always useful to review complications of anaesthesia but we must be wary of being wise after the event and making assumptions for which there is no evidence other than coincidence. Leigh and Tytler (*Anaesthesia* 1990; **45**:

814-20) report an apparently fit man who suffered brain death whilst under anaesthesia with induced hypotension, and describe the death as 'related to induced hypotension'. Keats wrote,<sup>1</sup> 'We are all brainwashed by the error-blame

mentality in reviewing anaesthetic records. . . . When brain damage follows an incident, the reviewer is much more likely to find inappropriate care—that is, errors—than if the patient recovers.'

Leigh and Tytler's patient had a lowest recorded systolic blood pressure of 90 mmHg. There will be many anaesthetic records in which blood pressures less than this, in sicker patients, caused no cerebral harm. Just because there was no other explanation for the event does not mean that an observation that *could* explain it *does* explain it.

A 76-year-old unpremedicated woman, who suffered from angina but to whom I had given an uneventful anaesthetic for surgery of the large bowel six weeks previously, arrested in asystole as I snapped open an ampoule of lignocaine before siting an intravenous cannula. No one would suggest the arrest was caused by opening the ampoule, but what if it had happened 3 minutes later, after I had given anaesthetic drugs? Consider another, rather more common, situation. A sick patient becomes hypotensive during anaesthesia and is later discovered to have suffered myocardial infarction. The anaesthetic record is reviewed and the anaesthetist chided for not treating the hypotension, the 'obvious' cause of the myocardial event. But what if the reverse were true, and the myocardial event had been the cause of the hypotension?

Association and causation are not the same, and doctors must be careful of ascribing causation—especially in

individual patients—no matter how plausible the connecting hypothesis.<sup>2</sup>

University Department of Anaesthesia, N.W. GOODMAN  
Medical School Unit,  
Southmead Hospital,  
Bristol BS10 5NB

#### References

1. KEATS AS. Anesthesia mortality in perspective. *Anesthesia and Analgesia* 1990; **71**: 113–9.
2. SKRABANEK P, MCCORMICK J. In: *Follies and fallacies in medicine*. Glasgow: Tarragon, 1989: 21–6.

#### A reply

Dr Goodman is wise to point out the existence of 'an error-blame mentality' which indeed will play a large part in all our futures.

In the case cited, with no other cause being found at autopsy, and in spite of a reasonably high intra-arterial blood pressure recorded continuously as a hard copy, HM Coroner sitting with an expert anaesthetist, came to the conclusion that there had been an 'Accidental Death' associated with the anaesthetic technique.

Royal Surrey County Hospital,  
Guildford GU2 5XX

J.M. LEIGH

#### Failure of the Ruben circle control valve

The control valve on a Ruben Circle anaesthesia system (AMBU: R. L. Dolby and Co) (Fig. 1) failed following the replacement of the valve at a routine service. The failure was caused by incorrect alignment of the 'mushroom' valve where it touches the plastic valve casing on the expiratory limb. This was noticed when the valve mechanism moved within its casing. Normally, on spontaneous inspiration the valve mushroom is drawn against the flange because the pressure within the mushroom is slightly higher than that in the expiratory limb. The faulty replacement valve did not make a perfect seal with the flange during spontaneous respiration because the mushroom had adopted an asymmetrical form. This resulted in rebreathing of exhaled gases in proportion to the fresh gas flow rate as described by Sik, Eveleigh and Lewis.<sup>1</sup> The arrangement of the unidirectional expiratory valve in the system prevents reverse flow from the reservoir bag through the absorber to the control valve. However, reverse flow through the

absorber is a normal feature of the flows within the system during late expiration and is caused by fresh gas entering a system which is full to its normal working volume. Such fresh gas then travels towards the spill valve flushing the expiratory limb. The fresh gas flow therefore dictates the volume of gas that can be inhaled from the expiratory limb in the presence of an incompetent flow valve. An important feature of this rebreathing is that the normal instinct for an anaesthetist on encountering rebreathing is to increase the fresh gas flow rate to flush the carbon dioxide out of the system. In the Ruben circle system, an increase in fresh gas flow in the presence of an incompetent control valve increases the rebreathing. There was sufficient pressure difference developed during intermittent positive pressure ventilation to push the mushroom onto the flange. Incompetence of the valve was only observed during spontaneous breathing. The workshop servicing tests applied on insertion of a new control valve do not detect

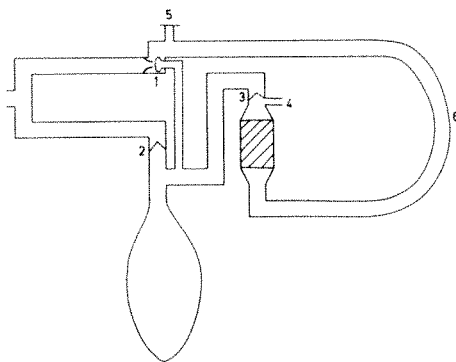


Fig. 1. The Ruben circle anaesthesia system. 1. Control valve. 2. Inspiratory valve. 3. Expiratory valve. 4. Fresh gas inlet. 5. Spill valve. 6. Expiratory reservoir limb.

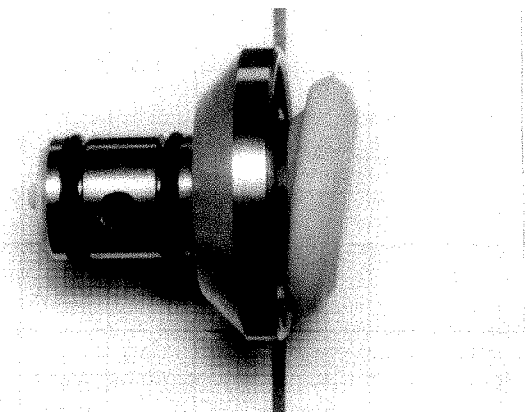


Fig. 2. The asymmetric set adopted by a tightly packaged Ruben circle control valve.

incompetence from this cause since the system is tested in IPPV mode only.

The reason for the valve failure was obvious when two further new valves were examined. The replacement valves are supplied in tight-fitting cardboard boxes lined with expanded foam pads. The mushroom is easily distorted by these pads allowing it to adopt an asymmetric configuration which persists for several weeks (Fig. 2). This serious packaging fault could be easily remedied by protecting the delicate mushroom control valve in transit by a removable dome. It is indeed regrettable that this otherwise excellent anaesthesia delivery system is jeopardized by such a simple and remediable oversight.

Royal Infirmary,  
Edinburgh EH3 9YW

M.R. LOGAN

#### Reference

1. SIK, MJ, EVELEIGH DJ, LEWIS RB. The Ruben circle anaesthesia system. An investigation of reverse flow of patient expired gas during spontaneous breathing. *Anaesthesia* 1988; **43**: 141-5.

#### A reply

This is the first time a packaging problem, as described by Dr Logan, has been reported to us. We will test the cardboard boxes and make sure the packaging is improved to avoid any recurrence.

Ambu International A/S,  
DK-2600 Glostrup,  
Denmark

J. SYDERS

#### Another complication of arterial cannulation

A 67-year-old female had a 20-gauge left radial artery cannula (Leader Cath) inserted during emergency abdominal aortic aneurysm repair. This was sutured *in situ* through each wing and also around the catheter barrel near the skin puncture site. The patient made an uneventful



Fig. 1. PA and lateral X rays of wrist showing cannula in the radial artery.

recovery and the intensive care staff were instructed to remove the line 3 days after the operation. The suture around the barrel was cut, as was the catheter barrel itself, the proximal part of which disappeared into the wrist. X ray findings are shown with the catheter visible within the radial artery. Full explanation was given to the patient and the line removed at arteriotomy under local anaesthesia with no further complications.

Complications of radial artery cannulation are widely documented,<sup>1,2</sup> including, infection, haematoma formation, thrombosis, thrombo-embolism, pseudo-aneurysm, air embolus and arteriovenous fistulae, but there are no reports of catheter loss. The use of a suture around the catheter barrel was contributory in the above case and is not recommended; sutures should only be necessary through the wings.

Derby City Hospital,  
Derby

M.M. BROWN

#### References

1. SLADEN A. Complications of invasive haemodynamic monitoring in the ITU. *Current Problems in Surgery* 1988; **XXV**: No 2.
2. FITZPATRICK GJ, MACDONALD NJ, MORIARTY DC. Radial artery cannulation—a prospective study. *Irish Journal of Medical Sciences* 1987; **156**: 279-81.

#### Acute renal failure in association with a pneumatic antishock garment and with tense ascites

Renal insufficiency associated with increased intra-abdominal pressure and relieved by abdominal decompression has been reported previously.<sup>1</sup> We wish to report a patient in whom this occurred on two occasions, each with a different cause. The patient was a 58-year-old man who underwent orthotopic liver transplantation for cirrhosis of uncertain aetiology. Before operation he was severely malnourished with grade 2 hepatic encephalopathy and noted to have tense ascites. His plasma urea was 6.1 mmol/litre and creatinine 115 mmol/litre. At surgery there was considerable haemorrhage from retroperitoneal tissues and in total 25 litres of blood was transfused. There was continued bleeding at the end of surgery and he was managed in a pneumatic antishock garment (PASG) (Gladiator Shock Suit) inflated to 30 mmHg.<sup>2</sup> During the operation urine output was in excess of 50 ml/hour and

immediately after the operation he passed 192 ml urine in the first 5 hours which was followed by only 59 ml in the next 24 hours; this was despite a central venous pressure in excess of 10 mmHg, mean arterial pressure in excess of 80 mmHg, renal dose dopamine, mannitol and high dose frusemide infusions. His haemodynamic variables during this period are summarised in Table 1. The PASG was progressively deflated over the second day to 10 mmHg. In the next 10 hours the urine output was 82 ml. The suit was then completely deflated and an immediate diuresis ensued; the patient passed 1783 ml in the next 14 hours.

No diuretics were given or alterations made to the rate of vasoactive drug infusions over this period and there was no significant change in measured haemodynamic variables which might have explained this diuresis. Plasma urea was 23.5 mmol/litre and creatinine 253 mmol/litre before his

**Table 1.** Haemodynamic variables before, during and after deflation of the PASG.

PASG pressure; mmHg	30	10	0
Mean arterial pressure; mmHg	75	95	80
Cardiac index	4.9	4.7	3.6
Systemic vascular resistance; dynes/second/cm <sup>-5</sup>	602	732	888
Oxygen delivery; ml/minute/m <sup>2</sup>	347	576	497
Oxygen consumption; ml/minute/m <sup>2</sup>	162	106	102
Urine output; ml/hour	3	8	127

diuresis which continued over the next 6 days, with a progressive decrease in his plasma urea to 18.1 mmol/litre and creatinine to 119 mmol/litre. Unfortunately his recovery was further complicated with recurrent sepsis, difficulties with weaning from ventilatory support and continuing encephalopathy. On the 22nd day after the operation he again became oliguric with no evidence of sepsis or haemodynamic disturbance, but by now he had developed tense ascites, massive scrotal oedema and peripheral oedema. His urine output did not respond to aggressive management with loop diuretics and mannitol and on day 24 it was decided to remove 2.5 litres of ascites. This resulted in immediate diuresis, urine output increasing from  $\approx 40$  ml/hour to greater than 140 ml/hour.

Richards and his colleagues reported 4 patients with anuria in association with increased intra-abdominal pressure from postoperative haemorrhage.<sup>1</sup> Polyuria and resolution of the renal failure occurred in each patient in response to operative decompression of the abdomen. In a dog model, pressures greater than 15 mmHg produced oliguria and when greater than 30 mmHg, anuria. Our patient remained oliguric even when the PASG had been deflated to 10 mmHg. The aetiology of acute renal failure associated with increased intra-abdominal pressure is not known; possibilities include an increase in renal venous pressure, a decrease in renal artery pressure and an increase in intravesical pressure leading to a reduction in the

perfusion pressure of the kidneys. Shenasky and Gillenwater<sup>3</sup> reported a decrease in urine output in dogs exposed to 30 mmHg counter pressure; however other workers appear to have found the detrimental effect of PASG application on urine flow to have diminished.<sup>4</sup> Roth and Rutherford<sup>5</sup> studied the effects of a PASG on regional blood flow during haemorrhagic shock and found improvement, even in areas encompassed by the suit. Haemodynamic effects following the release of increased intra-abdominal pressure have been well documented.<sup>6</sup>

We believe this patient emphasises the importance of increased intra-abdominal pressure on urine flow, which does not appear to be dependent on haemodynamic variables alone and also highlights a potential complication of the use of pneumatic antishock garments; oliguria was erroneously attributed to continuing haemorrhage.

Addenbrooke's Hospital,  
Cambridge CB2 2QQ

M.J. O'LEARY  
G.R. PARK

#### References

1. RICHARDS WO, SCOVILL W, SHIN B, REED W. Acute renal failure associated with increased intra-abdominal pressure. *Annals of Surgery* 1983; **197**: 183-7.
2. ABERG TW. Pneumatic antishock garments and intra-abdominal bleeding. Bulletin No. 71, Department of Surgery, Lund University, Sweden 1989.
3. SHENASKY JH, GILLENWATER JY. The renal hemodynamic and functional effects of external counterpressure. *Surgery, Gynecology and Obstetrics* 1972; **134**: 253-8.
4. CANGIANO JL, KEST L. Use of a G-suit for uncontrollable bleeding after percutaneous renal biopsy. *Journal of Urology* 1972; **107**: 360-1.
5. ROTH JA, RUTHERFORD RB. Regional blood flow effects of G-suit application during hemorrhagic shock. *Surgery, Gynecology and Obstetrics* 1971; **133**: 637-43.
6. SHELLY MP, ROBINSON AA, HESFORD JW, PARK GR. Haemodynamic effects following surgical release of increased intra-abdominal pressure. *British Journal of Anaesthesia* 1987; **59**: 800-5.

#### Cricoid pressure in the sniffing position

I was interested to read the letter from Drs Crowley and Giesecke (*Anaesthesia* 1990; **45**: 588-9) describing the evolution of their technique of bimanual cricoid pressure. They endorse the use of counter-pressure applied behind the neck as suggested by Crawford<sup>1</sup> and Gibbs and Modell<sup>2</sup> and I have no doubt that this improves the efficiency of the pressure on the cricoid cartilage in occluding the oesophagus. However, the head-neck position which they advocate is suboptimal for ease of intubation, since it does not align the three axes (oral, pharyngeal, and laryngeal)<sup>3,4</sup> necessary for visualisation of the larynx. It is interesting that Sellick<sup>5</sup> also described the extended neck position in his original description of cricoid pressure. Wraight *et al.*<sup>6</sup> found a significant intraluminal cricopharyngeal pressure in anaesthetised patients with the 'neck extended and head supported', but did not evaluate if this was related to position or to the other factors they considered i.e. 'compression by neighbouring structures or the configuration of the muscle components of the sphincter'.

I would suggest that when one is concerned about the potential for regurgitation and aspiration of stomach contents, then optimal intubation conditions should be sought. This necessitates the use of the full 'sniffing position' with the neck flexed on the shoulders and the head extended on the neck,<sup>3,4</sup> easily achieved by the use of a

small pillow beneath the head. The cervical lordosis is less exaggerated in this position compared with that of neck extension and cricoid pressure,<sup>7</sup> and is easily and effectively applied (with or without the bimanual technique).

Ottawa General Hospital,  
Ottawa, Ontario K1H 8L6

A.D. BAXTER

#### References

1. CRAWFORD JS. *Principles and practice of obstetric anaesthesia*, 5th edn. Oxford: Blackwell Scientific, 1984; 326-7.
2. GIBBS CP, MODELL JH. Cricoid pressure. In MILLER RD ed. *Anesthesia*, Vol. 3, 2nd edn. New York: Churchill Livingstone 1986: 2041.
3. STOELTING RK. In MILLER RD ed. *Anesthesia*, Vol. 1, 2nd edn. New York: Churchill Livingstone 1986: 525-8.
4. MURRIN KR. In LATTO IP, ROSEN M, eds. *Difficulties in tracheal intubation*. London: Bailliere Tindall, 1985: 75.
5. SELICK BA. Cricoid pressure to control regurgitation of stomach contents during induction of anaesthesia. *Lancet* 1961; **2**: 404-6.
6. WRAIGHT WJ, CHAMNEY AR, HOWELLS TH. The determination of an effective cricoid pressure. *Anaesthesia* 1983; **38**: 461-6.
7. LATTO IP. In: LATTO IP, ROSEN M, eds. *Difficulties in tracheal intubation*. London: Bailliere Tindall, 1985: 110-1.

### A complication of a flexible tracheostomy tube during laryngectomy

The Ruschelit flexible tracheostomy tube (Laryngoflex) is recommended for use during laryngectomy and is designed to prevent kinking during the suturing of the pharynx following removal of the larynx. We have recently encountered two serious cases of respiratory obstruction while using these tubes. In both instances, shortly after insertion of the tube, the peak airway pressure increased from 20 to 50 cmH<sub>2</sub>O, accompanied by decreased gas entry to both lungs, especially the right, with wheezing on auscultation. At the same time the arterial oxygen (SaO<sub>2</sub>) saturation decreased to 75%.

Bronchial intubation or cuff herniation was suspected. A suction catheter was passed through the tube without difficulty. The cuff was deflated and the tube withdrawn a short distance. Thereafter with each subsequent respiration, the cuff of the flexible tube displaced itself from the trachea, preventing fixation (Fig. 1). On both occasions we were forced to substitute the Ruschelit tube with the ordinary Portex tracheostomy tube. We believe that the obstruction was caused by the tip of the tube impinging on the carina or on the tracheal wall (Fig. 2). The distance between the cuff and the tip is very short and the fact that the tip is not bevelled further contributes to the development of obstruction. This probably caused a combination of a ball-valve mechanism maintained by the cuff and a rising intrathoracic pressure which quickly produced complete airway obstruction.

Similar cases of obstruction due to folding of a soft unprotected tip has also been reported by several authors

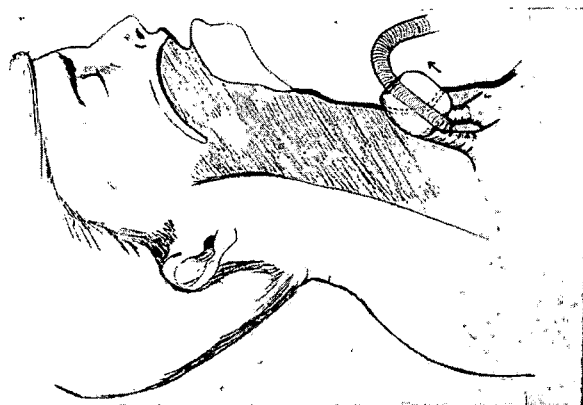


Fig. 1. The withdrawn tube displaced itself from the trachea, with each subsequent respiration, preventing fixation.

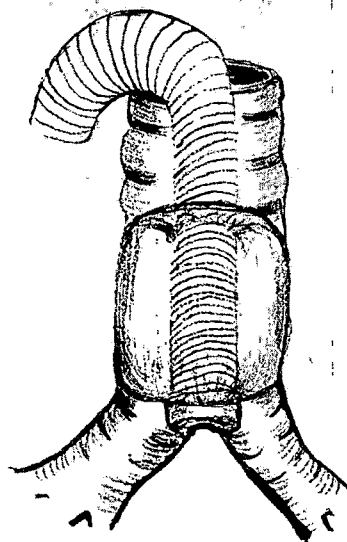


Fig. 2. The tip of the tube impinging on the carina causes airway obstruction.

using armoured tubes.<sup>1-3</sup> It is evident that the presence of an armoured tube cannot be regarded as a guarantee of a clear airway and that using such a tube for a laryngectomy could be a problem and even hazardous.

Kaplan Hospital,  
Rehovot 76100,  
Israel

T. EZRI  
D. HALPERIN  
S. LURIE  
D. SOROKER

### References

1. HEDDEN M, SMITH RBF, TORPEY BJ. A complication of metal spiral-imbedded latex endotracheal tubes. *Anesthesia and Analgesia* 1972; **51**: 859-62.
2. MIRAKHUR RK. Airway obstruction with cuffed armoured tracheal tubes. *Canadian Anaesthetists' Society Journal* 1974; **21**: 251-3.
3. NG TY, KIRIMLI NT, KIRIMLI BI. Hazards in use of anode endotracheal tube. A case report and review. *Anesthesia and Analgesia* 1975; **54**: 710-14.

### Sexual excitement following anaesthesia or sedation

Amorous and disinhibited behaviour following propofol anaesthesia has often been reported since its introduction.<sup>1,2</sup> This behaviour was initially thought to be amusing;<sup>3</sup> however, there have been recent accounts of distressing sexual fantasies following propofol anaesthesia and more commonly following sedation with benzodiazepines.<sup>2,4</sup>

I would agree strongly with recommendations of Drs Boheimer and Thomas (*Anaesthesia* 1990; **45**: 699) that a third party should be present when drugs which may alter

normal perception are administered, in view of the possibility of laying oneself open to allegations of sexual impropriety. However, I would question the advisability of forewarning patients of the possibility of sexual hallucinations or amorous behaviour. A major cause of pre-operative apprehension is the belief that the patient may do something embarrassing whilst under anaesthesia and a warning of this nature might well increase the anxiety of the patient. It is also possible that behaviour of this kind might be increased because of autosuggestion.



It is interesting to note that worries of sexual excitement following anaesthesia have been reported almost since the advent of anaesthesia. In the report of the Westminster Medical Society in the *Lancet* of 1849, G.T. Gream, Surgeon Accoucheur to Queen Charlotte's Lying-in Hospital was completely opposed to the use of chloroform, particularly in obstetrics.<sup>5</sup> He alluded to several cases in which women had, under the influence of chloroform, made use of obscene and disgusting language. Simpson, who was instrumental in the introduction of chloroform to anaesthetic practice, was prompted to reply to these allegations.<sup>6</sup> In a report from the Medico-Chirurgical Society of Edinburgh, also in 1849, he stated that chloroform had been in constant use in Edinburgh for 15 months without casualty and he had never heard of anyone having seen sexual excitement. He went on to say that 'After inhaling ether during her confinement in the Maternité, one Parisian prostitute stated that she had had lascivious dreams. But surely it was, to say the least, very unbecoming to say that most English ladies should have sexual dreams (like one French prostitute) when under the

influence of chloroform.' Are we very unbecoming to make such suggestions now?

Manchester Royal Infirmary,  
Oxford Road,  
Manchester M13 9WL

B.J. WOODCOCK

#### References

1. BRICKER SRW. Hallucinations after propofol. *Anaesthesia* 1988; **43**: 171.
2. DUNDEE JW. Sexual fantasies during midazolam sedation. *British Journal of Anaesthesia* 1990; **65**: 281P.
3. YOUNG PN. Hallucinations after propofol. *Anaesthesia* 1988; **43**: 170.
4. SCHAEFFER HG, MARSH SCU. An unusual emergence after total intravenous anaesthesia. *Anaesthesia* 1989; **44**: 928-9.
5. GREAM GT. Chloroform in midwifery. Report of the Westminster Medical Society. *Lancet* 1849; **1**: 99-100.
6. SIMPSON. Chloroform in maternity. Report from the Medico-Chirurgical Society of Edinburgh. *Lancet* 1849; **1**: 235-6.

#### The cost of propofol infusion in neurosurgery

We read with interest correspondence on the cost of propofol relative to other agents in day case surgery,<sup>1,2</sup> since we have been examining this problem in another context. Propofol administered as a continuous infusion may be preferred to conventional inhalational techniques in prolonged surgery on account of the rapidity of recovery from anaesthesia.<sup>3</sup> However, as previous correspondents have noted, it is a relatively expensive agent.

We examined the cost of maintenance of anaesthesia in 24 patients undergoing semi-elective clipping of intracranial aneurysms. Eleven patients were maintained with a nitrous oxide-oxygen-isoflurane technique, and 13 with oxygen-air-propofol. In both groups depth of anaesthesia was assessed on clinical grounds, i.e. heart rate, blood pressure, sweating, and the isoflurane or propofol adjusted accordingly.

Costing was done as follows: propofol technique: number of ampoules opened counted, start and end of infusion times noted; inhalational technique: isoflurane vaporizer filled to line before start, and refilled to line at end; volume of isoflurane used measured to nearest 10 ml; nitrous oxide maintained at steady flow throughout; start and end times noted. The costs of these agents were supplied by our hospital pharmacy and are as follows: propofol, 5 × 20 ml ampoules, £16.50; nitrous oxide, 5000 litre cylinder, £20.32; isoflurane, 100 ml, £32.50. Costs which were common to both groups such as muscle relaxants, antibiotics and analgesics were not included, nor was the cost of the induction agent.

In the inhalational group, the mean duration was 252 minutes (range 165-320) with a cumulative duration of 2780 minutes. This resulted in a total cost of £269.20 or

£5.81/hour. The corresponding figures in the propofol group were 291 minutes (range 205-370), 3787 minutes, £648.50 and £10.27/hour.

There was a degree of interindividual variability in both groups, as might be expected. Costs in individual patients ranged from 5.7p/minute to 17p/minute in the inhalational group, and from 12.1p/minute to 25.7p/minute in the infusion group.

Thus it can be seen that propofol infusion is markedly more expensive than an inhalational technique using isoflurane, which, while it is the volatile agent of choice in neuroanaesthesia, is the most expensive currently available. Use of a circle system would render the inhalational technique even less expensive. However, relative to the total cost of the patient's stay in hospital (for instance, the Sugita clips used during these operations cost £80-£100 each) we suggest the difference is not so great as to be a major factor influencing choice of anaesthetic technique.

Institute of Neurological Sciences,  
Southern General Hospital,  
Glasgow G51 4TF

H.A. AITKEN  
P.A. FARLING

#### References

1. McWILLIAM J. The costs of propofol in day surgery. *Anaesthesia* 1989; **44**: 866.
2. SCOTT PV. The costs of propofol in day surgery. *Anaesthesia* 1990; **45**: 174-5.
3. HARTUNG E, FREYE E. An open comparison of propofol and enflurane for prolonged abdominal operations. *Anaesthesia* 1988; **43** (suppl): 105-7.

#### Regional anaesthesia must be properly managed

Dr Wildsmith and Professor Aitkenhead are to be commended on their correspondence (*Anaesthesia* 1990; **45**: 984-5); they no doubt subscribe, as I have since the late 1960s, in principle, to the content of the statement made by Dr Daniel C. Moore in 1982 at the first European Society of Regional Anaesthesia Meeting in Edinburgh, 'to

perform (most forms of) regional anaesthesia without sedation shows crass disregard for patient comfort and well-being'. An explanation of possible subjective feelings during a procedure should be made to a patient without causing anxiety. The old adage of a pre-operative visit is as good as or better than premedication alone still stands.

But their discussion of sedation is somewhat unclear. Conscious sedation with preservation of reflexes and continuous verbal contact, a situation easily maintained by careful intravenous titration of one's patient is ideal and subjective feed-back reliable. The patient who is unhappy for any reason at all can say so and the position rectified to their satisfaction. Amnesia can be ensured and unconsciousness induced if it is their wish. If

unconsciousness from the outset is the patient's wish it is to be respected and that is not sedation, by definition.

Explanation beforehand and agreement reached with one's patient is surely truly informed consent.

3 Carnoustie Drive,  
Macclesfield SK10 2TB

R.H. DALE

### Suxamethonium and intensive care

I was intrigued to read the case reported by Dr Hemming and colleagues of severe hyperkalaemia causing cardiac arrest following intravenous suxamethonium and propofol (*Anaesthesia* 1990; 45: 990-1). I am aware of at least one similar case with, unfortunately, a fatal outcome.

I note that the authors considered the possibility of denervation, but concluded that the hyperkalaemia was due to suxamethonium-induced release of potassium from muscle affected by disuse atrophy. It has long been thought that the weakness occurring in critically ill patients was due to a combination of immobility and malnutrition, together with the effects of drugs such as steroids and muscle relaxants. Zochodne and co-workers,<sup>1</sup> however, investigated 19 patients who presented with weakness as a complication of multiple organ failure and sepsis. Physical signs were variable, but investigation revealed electromyographic changes compatible with denervation together with axonal degeneration on neurophysiological testing or postmortem examination. It has been suggested that this complication may occur in as many as 50% of

ITU patients after 2 weeks, and the onset may be as early as six days after intubation. Dr Hemming's patient had been on ITU for 34 days at the time of the incident and was obviously weak at the time of discharge. I suspect that the patient was suffering from 'critical illness neuropathy' in which muscle denervation could lead to hyperkalaemia following administration of depolarising muscle relaxants.

The lesson is that suxamethonium should be used with great caution in critically ill patients, particularly in prolonged illness and that denervation cannot be excluded by clinical examination alone.

St. Bartholomew's Hospital,  
London EC1A 7BE

J. COAKLEY

### Reference

1. ZOCHODNE DW, BOLTON CF, WELLS GA, GILBERT JJ, HAHN AF, BROWN JD, SIBBALD WA. Critical illness polyneuropathy. A complication of sepsis and multiple organ failure. *Brain* 1987; 110: 819-42.

### Insertion of laryngeal mask airway in children

The laryngeal mask airway (LMA) is a useful addition to paediatric anaesthetic practice. Mason and Bingham reported their experiences with paediatric LMAs (*Anaesthesia* 1990; 45: 760-3) and concluded that it was an additional useful aid in the management of children during spontaneous respiration. My own experience of using this airway is very similar to theirs. In an endoscopic review of cases where only the size 2 LMA was available I found that the epiglottis was folded down over the vocal cords in 32 patients, despite there being a clear airway, a good seal and no gastric distension by the anaesthetic gases.

The reason for this is, I believe, that when the cuff is inflated the tongue is pushed anteriorly thus raising it and the epiglottis. In 24 children I had to use the manoeuvre of

twisting the LMA when it met the resistance of the posterior pharyngeal wall, which is set at more of an acute angle to the floor of the mouth than in adults, and this resulted in successful insertion, but since this study, it has now become my practice to insert the LMA 'back to front' i.e. with the black line pointing towards the lower teeth and twisting it as the resistance of the posterior pharyngeal wall is felt. This is similar to the method of inserting a Guedel airway, and as it seems to have been rapidly accepted by other members of our division as the standard method of insertion it is one that I heartily recommend.

Royal Hospital for Sick Children,  
Glasgow G3 8SJ

L.R. McNICOL

### Race and Apgar scores

The authors of the publication Race and Apgar scores (*Anaesthesia* 1990; 45: 988-9) used a memorial to Virginia Apgar as their reference.<sup>1</sup> Had they availed themselves of the original,<sup>2</sup> they would have noted that Dr Apgar realized colour to be 'the most unsatisfactory sign.' 'The foreign material so often covering the skin of the infant at birth interfered with interpreting this sign, as did the inherited pigmentation of colored children.' Some years later, Crawford and his coworkers<sup>3</sup> analysed the significance of the individual components of the Apgar score and

concluded that 'inclusion of the score for colour reduces the discriminatory value of the total score.' They recommended that 'in clinical practice as well as in reports, reference be made to the Apgar-minus-colour score.' Crawford's data were confirmed in a prospective study<sup>4</sup> of 66 unselected neonates which revealed that 'exclusion of the score for colour increases the correlation between the total score and umbilical artery blood biochemical data.'

Rather than accepting that 'the optimal Apgar score for an Afro-American infant should be therefore lower', colour

should be eliminated as a component of the Apgar score of all infants.

Albert Einstein College of Medicine,  
The Bronx,  
New York 10461, USA

A.Z. YAMA  
G.F. MARX

#### References

1. BECK J. Virginia Apgar, in memoriam. In ALADJEM S, BROWN A, SUREAU C, eds. *Clinical perinatology*, 2nd edn. St. Louis: C.V. Mosby, 1980: 19-29.
2. APGAR V. A proposal for a new method of evaluation of the newborn infant. *Current Researches in Anesthesia and Analgesia* 1953; **32**: 260-7.
3. CRAWFORD JS, DAVIES P, PEARSON JF. Significance of the individual components of the Apgar score. *British Journal of Anaesthesia* 1973; **45**: 148-58.
4. MARX GF, MAHAJAN S, MICLAT MN. Correlation of biochemical data with Apgar scores at birth and at one minute. *British Journal of Anaesthesia* 1977; **49**: 831-3.

#### The Rex Binning Department of Anaesthesia

Many of your readers will remember with great affection and respect Dr Rex Binning, who died 2 years ago after celebrating his 80th birthday in typical style. They will be pleased to know that the Brighton department became officially known as The Rex Binning Department of Anaesthesia on 31 October 1990. Geraldine his widow and Peter his son were present alongside a number of Rex's former colleagues and members of the Department.

Rex Binning's contribution to anaesthesia in general was extensive and to anaesthesia in Brighton was unique. He saw what was to come long before most people. During the 1960s and early 1970s Rex had set up senior registrar and registrar rotations with King's and Guy's and insisted on proper accommodation and secretarial support for the Department. Study leave and attendance at courses for the examinations was normal for Brighton trainees when still the exception elsewhere. Rex was, of course, a regular contributor to *Anaesthesia*. He travelled widely and his name was everywhere linked with Brighton anaesthesia. The trainees who came under Rex Binning's influence are scattered around the world. The members of today's Department trust that they will approve of this gesture in his memory.

15 Brangwyn Avenue,  
Brighton BN1 8XH

P.A.D. WILLIAMS



Fig. 1. Rex Binning.

#### Nudging the emergency oxygen

It was noticed after a clinical incident that the emergency oxygen button on a Boyle International Mark II can be inadvertently pressed and indeed locked by the proximal valve block of a Bain coaxial system. A clockwise twist can be imparted when pressed against the oxygen button, enough to lock the button (Fig. 1). The increased flow of gas is not easily noticed when using a Bain system with a Nuffield Penlon ventilator.

Kettering General Hospital,  
Northamptonshire NN16 8UZ

Z. HANAFIAH  
W.F.S. SELLERS



Fig. 1.

## Book reviews

<b>Pharmacokinetics</b>	332	<b>Manual of medical care of the surgical patient</b>	333
S.B. HLADKY		Edited by T.R. COUSSONS, P.A. MCKEE AND G.R. WILLIAMS	
<b>Clinical neuroanaesthesia</b>	332	<b>Books received</b>	334
Edited by R.F. CUCCHIARA AND J.D. MICHENFELDER			
<b>Introduction to intensive care</b>	333		
Edited by D. POTTER			

### Pharmacokinetics

S.B. HLADKY. Pp. x + 157. Manchester University Press, 1990. £6.95.

Pharmacokinetics, the description of the processes that determine the concentrations of a drug and its metabolites within the body, appears to be an increasingly accurate but complex science. Anaesthetists need a clear guide as to the terminology and applications of this branch of knowledge. Dr Hladky provides one. His small book is divided into four sections. The first introduces the necessary definitions and ties these to the associated physiological processes wherever possible. Thus it covers areas such as clearance, renal excretion, drug metabolism, hepatic elimination and routes of absorption of drugs. The second part shows how these factors can be used to determine the time course of a drug after an intravenous bolus, an intravenous infusion or after absorption via another route — such as an oral dose. The patterns to be seen after multiple dosing are also assessed. There is one chapter describing what happens with thiopentone and with volatile anaesthetics which shows how more complex models are often necessary. The third section deals with more advanced topics including the problems associated with plasma protein binding and with the methods used to assess the parameters of various models once one knows the time course of the plasma concentration. The final part includes some worked examples.

There is a lot in the book. The reader will only gain from it as much as he (she) puts in; this is one of the books which needs to be read 'wholly and with diligence and attention' as Bacon would suggest. It will complement any lecture course on pharmacokinetics and is a remarkably cheap addition to the literature.

J. NORMAN

### Clinical neuroanaesthesia

Edited by R.F. CUCCHIARA AND J.D. MICHENFELDER. Pp. xii + 555. Churchill Livingstone, 1990. £57.50.

It is my opinion that the most enjoyable parts of scientific meetings, and arguably the most useful, are the times spent between the formal sessions (the titles of which promise much and frequently deliver little) talking to people with similar problems to oneself. On the one hand, a certain degree of pride is experienced that one has changed attitudes or techniques rendering the problem insignificant, mingled with astonishment that everyone is not doing it

this way, on the other, receiving information, in retrospect often quite obvious, that will be put into practice on the next available theatre list. Reading this book is a rather similar experience. The authors of the individual chapters are all current or erstwhile members of the staff at the Mayo Clinic and their avowed aim, as stated in the preface, is to say 'This is the way we do it at the Mayo Clinic'. Fascinatingly, the preface also contains the workload for the neurological centre at the Clinic, together with an indication of staffing ratios, providing UK units with a yardstick whereby they may judge their own performances.

The book is divided into the usual two parts for texts on neuroanaesthesia, Scientific Foundations and Clinical Applications. Dr Michenfelder tackles cerebral blood flow etc. in the first part together with chapters on CSF dynamics, intracranial pressure, neurological monitoring and, usefully, an update on cerebral protection. The second part contains reviews on the anaesthetist's role in various types of neurological procedures together with neurointensive care, acute and chronic pain and brain death.

The level of didacticism varies from chapter to chapter. Some authors sit on the fence presenting a review of the evidence but without a definite statement 'This is how I do it'; others put up their technique to be sniped at — much more fun. Some techniques are technically far advanced from those in our centre; some we would consider unnecessarily primitive. The drugs described do not coincide with those available in the UK, as might be expected and their use would be considered inappropriate in some centres; we would not now consider deliberate hypotension during the routine clipping of aneurysms of the circle of Willis. None of this implies criticism of the book, rather it reflects my disappointment at not being able to turn monologue into dialogue and the frustration of crying 'Rubbish!' to a printed page.

The illustrations are few but appropriate, the index is adequate and the references excellent. One of the major uses of this type of book is to allow the reader access to relevant literature without having to trawl back through the *Index medicus*. A minor criticism is that the references are somewhat parochial; a view frequently promulgated by those of jaundiced view who are unable to find their own work cited.

In summary, this book should be read, as intended by the editors, with discretion; it is not a recipe book for beginners. Its price may put off many from buying it individually, but it would form a most useful addition to a departmental library. Its authors should consider frequent revisions in order to provide a continuing source of updated references. My major worry is that the neurosurgeon with (I almost wrote 'for') whom I work has donated a copy of this book to the departmental library. His

reason? The views expressed on 'anaesthesia in the sitting position', are rather less vitriolic than those of his neuro-anaesthetic colleagues.

D. SAUNDERS

### Introduction to intensive care

Edited by D. POTTER. Pp. 472 + Index. Farrand Press/Portland Press, 1990. £38.80.

This multi-author book, using medical expertise associated mostly with the King's College School of Medicine, purports to provide a structured introduction for the newcomer to intensive care and to endeavour to whet his (her) appetite for further study. This book should make a good impression on junior doctors because it is mostly a practical, easily readable and concise treatise packed with many useful tables and algorithms. There are many excellent chapters, such as those on the cardiovascular system, and I especially appreciated the inclusion of a chapter on acute metabolic emergencies.

This overall favourable impression is regrettably marred, firstly by some notable omissions. An important advance in intensive care which has occurred in the past 5 years, is our ability to stratify critically ill patients by means of severity of disease-scoring systems. This aspect receives no mention. There is also, remarkably, no mention in the chapters on respiratory failure and the technique of respiratory support, of the use of continuous arterial saturation, or in the book as a whole, of mixed venous oxygen saturation, which are generally recognised as major advances in intensive care monitoring. There is no mention in the discussion on pulmonary embolism of the usefulness of end-tidal CO<sub>2</sub> monitoring; a sudden decrease in the end-tidal CO<sub>2</sub> can alert one to this diagnosis. An important topic, which receives only very cursory mention, is the use of selective decontamination of the gut to reduce nosocomial infection in critically ill patients whose lungs are being ventilated. The advice that the prophylactic use of antacids and H<sub>2</sub> receptor antagonists against haemorrhage in patients receiving artificial ventilation is to be recommended, must now be challenged. Present evidence suggests that antacids and H<sub>2</sub> receptor antagonists do not protect against bleeding in the upper gastrointestinal tract and that there are better and safer ways of protecting against this form of haemorrhage. The disadvantage of changing the gastric contents to an alkaline milieu is the overgrowth of Gram-negative bacteria and its association with Gram-negative nosocomial pneumonias. Althesin is recommended as one sedative drug for consideration in severe head injuries. There is no doubt that it was a very useful drug for sedation in patients undergoing artificial ventilation with severe head injuries, but it has been out of production now for several years! The currently popular sedative drug, propofol, is not discussed. There is no mention of the importance of optimising the serum sodium concentration in head-injured patients. It is hoped that no doctor takes the advice on page 234 of adding 510 Iu of insulin to a litre of peritoneal dialysate fluid if hyperglycaemia occurs!

Other criticisms relate to typographical errors: several words in the text and in the tables are misspelt, but more seriously, inaccuracies involving some numbers and units occur. Sub- and superscripting were occasionally absent making several units look confusing. The ECG depicted in Figure 2 on page 62 is presented upside down! There is frustratingly confusion over the greater (>) and less than (<) signs in several places and these are even omitted

altogether in several instances. Punctuation is deficient in several places. Although many of the diagrams and tables are excellent, some give the appearance of being amateurish and in a few cases are presented in exceedingly small print. Referencing in a few instances is inaccurate. An entire sentence appears to have been misplaced in one paragraph on page 239. Some of the Figures have no explanations for their abbreviations. Much of the criticism could have been avoided by more careful proof reading. The revised edition should create a more professional impression.

S. JACOBS

### Manual of medical care of the surgical patient

Edited by T.R. COUSSONS, P.A. MCKEE AND G.R. WILLIAMS. Pp. xiii + 324. Little, Brown, 1990. \$24.50.

This is the fourth edition of a manual which has been edited by Professors from the departments of Medicine and Surgery of the University of Oklahoma College of Medicine, with contributions from Professors in the departments of Anesthesiology, Medicine and Surgery. It is written for physicians involved in the care of patients undergoing surgery and is a handy size, presumably to fit into a white coat pocket. The spiral binding with loose leaf pages is useful, but these are easy to damage and produce confetti. The main aim is stated to be the detection and management of medical conditions encountered in pre-, intra- and postoperative patients with priority given to more common problems.

The first chapter briefly reviews various techniques and agents used in anaesthetics, the pre-operative assessment and basic monitoring. Thereafter the chapters relate to organ systems not including paediatrics, the special senses or the reproductive system, followed by separate chapters on the elderly, the patient with AIDS and trauma patients. The format is designed for fast reference and the index is fairly comprehensive, but there are omissions in the latter e.g. pacemaker (although this is covered in the section on cardiac arrhythmias), oxygen saturation which is listed in the first chapter and intercostal nerve block which is mentioned as a method of pain relief after thoracotomy. Pneumothorax cannot be found either in the chapter on the respiratory system or on trauma.

One of the stated emphases in the manual is on the detection and management of medical problems with assessment of risk. It would be expected that common postoperative complications would be listed with an indication of their incidence, but this is not so. The detection of postoperative hypoxaemia and the rational use of oxygen therapy in the postoperative period is not discussed. The section on postoperative management of pulmonary complications describes general therapeutic manoeuvres, such as the choice of analgesics, a 'stir up' regimen of early ambulation, coughing, incentive spirometry, intermittent positive pressure breathing, continuous positive pressure (of what gas?) applied by facemask, humidification and chest physiotherapy. For specific complications such as progressive atelectasis, maximum yawning is first advised with chest physiotherapy and bronchoscopy if the aetiology is a mucus plug. Perhaps the use of oxygen is *prima facie*, nevertheless, students and practitioners involved in surgical patient care should be taught that oxygen is required in the immediate postoperative period and this use may be extended in particular patients without waiting for signs of frank respiratory failure to develop. Anaesthetists who are looking for a medical text to help them in preparation for

the Fellowship examinations may find difficulties with this manual. Measurements are not made in SI units, and the book is limited by its brevity and the omission of topics which are covered in general medical text books for students. It is published for the faculty, house staff and students at the University of Oklahoma College of Medicine and is obviously popular since it has been revised frequently since 1976, but for the trainee anaesthetist in the United Kingdom it is of limited value.

A. HOLDCROFT

#### Books received

We thank the publishers for the following, books, some of which may be reviewed in future issues of *Anaesthesia*.

#### **Anesthesia for vascular surgery**

Edited by M.F. ROIZEN. Pp. xvii + 505. Churchill Livingstone, 1990. £55.00

#### **Clinical applications of ventilatory support**

Edited by R.R. KIRBY, M.J. BANNER AND J.B. DOWNS. Pp. xii + 546. Churchill Livingstone, 1990. £49.95.

#### **Advances in anesthesia, vol 7**

Edited by R.K. STOELTING, P.G. BARASH AND T.J. GALLAGHER. Year Book Medical, 1990.

#### **Year book of critical care medicine 1990, 8th edn.**

Edited by M.C. ROGERS AND J.E. PARRILLO. Pp. xvii + 338. Year Book Medical Publishers, 1990. £41.50.

#### **Case studies in critical care medicine, 2nd edn.**

Edited by R.D. CANE, B.A. SHAPIRO, R. DAVISON. Pp. xii + 444. Year Book Medical, 1990. £34.00.

#### **An introduction to cardiovascular physiology**

Edited by J.R. LEVICK. Pp. vii + 279. Butterworths, 1990. £14.95.

#### **Baillière's Clinical Anaesthesiology**

#### **International Practice and Research.**

#### **Anesthesia for Day Case Surgery. Vol. 4.**

Edited by T.E.J. HEALY. Baillière Tindall, 1990. £22.50.



## Obituaries

- Bodley, P.O.**, TD, FCAnaes, DCH, DOBst RCOG. Formerly Consultant Anaesthetist, Oldchurch Hospital, Romford. Qualified from University of London, 1953.
- Hain, W.R.**, MB, BS, FCAnaes. Formerly Consultant Paediatric Anaesthetist, University Hospital and City Hospital, Nottingham. Qualified from University of London, 1962.
- Leishman, W.J.**, MB, ChB, DA, DOBst RCOG. Formerly Assistant Anaesthetist Stirling Royal Infirmary, Stirling. Qualified from Glasgow University 1954.
- Welpley, N.C.**, MBE, MRCS, LRCP, DA. Formerly Consultant Anaesthetist Stoke Mandeville Hospital. Qualified from University of London 1942.
- Young, H.S.A.**, FFARCSI. Formerly Consultant Anaesthetist, Royal Victoria Hospital, Belfast. Qualified from Queen's University Belfast 1968.

## International congress calendar

1991

- 4–7 April.** Cincinnati. *16th Annual Meeting of the American Society of Regional Anesthesia*.  
Information: P.O. Box 11086, Richmond, Virginia 23230–1086, USA.
- 3–5 April.** Oxford. *Junior Anaesthetists' Group of the Association of Anaesthetists of Great Britain and Ireland Linkman Conference and Annual Scientific Meeting*.  
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 11–13 April.** Belgium. *Update in Cardiac Surgery, Anaesthesia and Intensive Care*.  
Information: The Secretary, Department of Anesthesia B11-5 de Pintelaan 185, B9000 Gent, Belgium.
- 15–18 April.** Hamamatsu, Japan. *6th International Symposium on Computing in Anesthesia and Intensive Care*.  
Information: Dr K. Ikeda, Chairman of the Organising Committee, c/o Department of Anesthesiology, Hamamatsu University School of Medicine, 3600 Handa-cho, Hamamatsu, Shizuoka, 431–31 Japan.
- 17–21 April.** Antilles. *19th International Society on Oxygen Transport to Tissue*.  
Information: Professor W. Erdmann, Department of Anaesthesiology, Dr Molewaterplein 40, 3015 GD Rotterdam, The Netherlands.
- 18–20 April.** Paris. *European Academy Open Scientific Meeting*.  
Information: Professor J.C. Otteni, Service d'anesthésie et de réanimation, Hôpital de Hautepierre, Avenue Molière, F-67098 Strasbourg, France.
- 18–21 April.** Paris. *European Academy of Anaesthesiology. Refresher Course*.  
Information: Professor J.M. Desmonts, Département d'Anesthésie, Hôpital Bichat, 46 rue Henri-Huchard, 75018 Paris, France.
- 23–27 April.** Montreal. *Second International Symposium on Pediatric Pain*.  
Information: Pain Secretariat, 3450 University Street, Montreal, Quebec, H3A 2A7, Canada.
- 3–5 May.** Philadelphia. *AUA Annual Meeting*.  
Information: Stephen J. Prevoznik, Department of Anesthesia, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, Pennsylvania 19104, USA.
- 4–8 May.** San Antonio. *Society of Cardiovascular Anesthetists*.  
Information: P.O. Box 11086, Richmond, Virginia 23230–1086, USA.
- 5–12 May.** Sydney. *Faculty of Anaesthetists, R.A.C.S., General Scientific Meeting*.  
Information: Faculty of Anaesthetists, Royal Australasian College of Surgeons, Spring Street, Melbourne 3000, Australia.
- 9–12 May.** Washington DC. *6th International Dental Congress on Modern Pain Control*.  
Information: American Dental Society of Anesthesiology, Inc., 211 E. Chicago Avenue, Suite 948, Chicago, IL 60611, USA.
- 16–19 May.** California. *Californian Society of Anesthesiologists Annual Meeting*.  
Information: Californian Society of Anesthesiologists, 1065 East Hillsdale Boulevard, Suite 410, Foster City, California 94494, USA.
- 18–19 May.** Taiwan. *1st Asian-Oceanic Symposium on Regional Anaesthesia*.  
Information: Professor J.H. Lee, P.O. Box 26-473 Taipei, Taiwan 10713.
- 24–25 May.** The Netherlands. *Receptors of the Brain, Lung and Heart: State of the Art*.  
Information: Cader Research B.V., P.O. Box 85, 4854 ZH Breda/Bavel, The Netherlands.
- 27–29 May.** Innsbruck. *Fourth International Symposium on Echocardiography and Doppler in Cardiac Surgery*. Hotel Scandic Crow, Innsbruck, Austria.  
Information: G. Manner, MD, c/o Inter convention, A-1450 Vienna, Austria.
- 27–28 May.** Paris. *International Symposium on the Management of Acute and Cancer Pain*.  
Information: Dr Claude Sainte-Maurice, Department of Anaesthesia, Université Descartes, Hôpital St Vincent de Paul, Paris, France.
- 27–31 May.** Montreal. *McGill University Annual Review Course in Anaesthesia*.  
Information: Post Graduate Board, Royal Victoria Hospital, 687 Pine Avenue West, Room H308, Montreal Quebec, H3A 1A1, Canada.
- 1–2 June.** Texas. *Eighth Annual Pain Symposium*.  
Information: James E. Heavner, DVM, PhD, Department of Anaesthesiology, Texas Tech University Health Sciences Center, 3601 Fourth Street, Lubbock, Texas 79430, USA.
- 4–7 June.** Milano. *6th Annual Meeting of the European Association of Cardiothoracic Anaesthesiologists*.  
Information: Francesca Rovelli, The Organizing Secretariat O.I.C. Incentive, Viale Majno, 21, 20122 Milano, Italy.
- 13–16 June.** Florida. *Florida Society of Anesthesiologists Annual Meeting*.  
Information: Florida Society of Anesthesiologists, 3000 34th Street South, Suite F, St. Petersburg, Florida 33711, USA.
- 21–25 June.** Quebec City. *48th Annual Meeting of Canadian Anaesthetists' Society*.  
Information: Ms Ann Andrews, CAS, 187 Gerrard St. E., Toronto, Ontario M5A 2E5, Canada.
- 24–28 June.** Norway. *21st Congress of the Scandinavian Society of Anaesthesiologists*.  
Information: Department of Continuing Education, The Norwegian Institute of Technology, N-7034 Trondheim, Norway.
- 21–23 August.** Edinburgh. *Edinburgh Anaesthesia Festival*.  
Information: Dr A.J. Pollock, Department of Anaesthetics, Royal Infirmary, Edinburgh EH3 9YW.
- 22–24 August.** Auckland. *The Annual Conference of New Zealand Anaesthetists*.  
Information: Department of Anaesthesia, Auckland Hospital, Park Road, Auckland, New Zealand.
- 25–31 August.** Willemstad, Curacao, Netherlands Antilles. *19th Meeting of the International Society of Oxygen Transport to Tissue (ISOTT)*.  
Information: Mrs Denise Haas, Department of Anaesthesiology, Erasmus University, Dr Molewaterplein 40, 3015 GD Rotterdam, The Netherlands.
- 28 August–1 September.** Strasbourg. *European Academy Scientific Meeting*.

- Information: Professor J.C. Otteni, Service d'anesthésie et de Réanimation, Hôpital de Haute-pierre, Avenue Molière, F-67098 Strasbourg, France.
- 4-8 September.** Rio de Janeiro. *XXI Latin American Congress of Anaesthesiology (WESA)*.  
Information: Dra M.B. de Azevedo, Rua Paulo Barreto 60, Botafogo, CEP 22280 Botafogo, Rio de Janeiro, RJ, Brazil.
- 6-8 September.** Texas. *Texas Society of Anesthesiologists*.  
Information: Texas Society of Anesthesiologists, 1905 North Lamar Boulevard, # 107, Austin, Texas 78705, USA.
- 11-13 September.** Harrogate. *Linkman and Annual Scientific Meeting of Association of Anaesthetists of Great Britain and Ireland*.  
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 17-21 September.** Patras, Greece. *9th Greek Congress of Anaesthesiology, Intensive Care and Emergency Medicine*.  
Information: Prof. C. Alexopoulos, Department of Anaesthesiology, Medical School, King George Square 30, Patras 262 21-Greece.
- 21-23 September.** Tokyo, Japan. *Third World Congress on Sleep Apnea and Rhonchopathy (III WCSAR)*.  
Information: Secretariat for III WCSAR, Simul International Inc., Kowa Building, No. 9, 1-8-10 Akasaka, Minato-ku, Tokyo 107, Japan.
- 11-15 October.** Baghdad. *4th Pan-Arab Congress of Anaesthesia and Intensive Care*.  
Information: Dr M. Keilani, P.O. Box 17078, Amman, Jordan.
- 26-30 October.** San Francisco. *American Society of Anesthesiologists Annual Meeting*.  
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 6-9 November.** Kuala Lumpur. *7th Asian Congress of Anaesthesiologists*.  
Information: Dr S.W. Lim, Pantai Medical Centre, 59199 Kuala Lumpur, Malaysia.
- 8-10 November.** Vina del Mar. *2nd Congress of Fed. of South American Soc. of Anesthesiologists*.  
Information: Dr Guillermo Lema, Av. Providencia 1476 (Depto. 405) Santiago, Chile.
- 8-11 November.** Toronto. *Paediatric Anaesthesia Conference*.  
Information: Sheila M. Peart, Paediatric Anaesthesia Conference, The Hospital for Sick Children, 555 University Avenue, Toronto, Ont M5G 1X8.
- 1-4 December.** Bangkok. *6th Congress of Western Pacific Association of Critical Care Medicine*.  
Information: Dr P. Sakolsatayadorn, Surgery, Siriraj Hospital, Bangkok 10700, Thailand.
- 6-8 December.** Washington. *Washington State Society of Anesthesiologists Annual Meeting*.  
Information: Washington State Society of Anesthesiologists, 2033 Sixth Avenue, # 804, Seattle, Washington 98121, USA.
- 7-11 December.** New York. *Forty-fifth Postgraduate Assembly in Anesthesiology*.  
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

## 1992

- 1-8 February.** Colorado. *18th Annual Vail Conference in Anaesthesiology*.  
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101, USA.
- 13-17 March.** San Francisco. *66th Congress of the International Anesthesia Research Society*.  
Information: International Anesthesia Research Society, 3645 Warrenville Center Road, Cleveland, Ohio 44122, USA.
- 25-29 March.** Tampa. *17th Annual Meeting of the American Society of Regional Anesthesia*.  
Information: P.O. Box 11086, Richmond, Virginia, 23230-1086, USA.
- 29 March-2 April.** Atlanta, Georgia. *The Third International Symposium on the History of Anaesthesia*.  
Information: R.K. Calverley, Medical Center, University of California, 225 Dickinson Street, San Diego, California CA 92103, USA.

- 1-3 April.** Bristol. *Junior Anaesthetists' Group Linkman Conference and Annual Scientific Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 2-6 May.** Boston. *Society of Cardiovascular Anesthetists*.  
Information: P.O. Box 11086, Richmond, Virginia, 23230-1086, USA.
- 4-9 June.** Toronto. *49th Annual Meeting of the Canadian Anaesthetists' Society*.  
Information: 187 Gerrard Street E, Toronto, Canada M5A 2E5.
- 7-12 June.** Barcelona. *Anesthesia 92*.  
Information: Pacifico, S.A.: c/Muntaner, 112 08036-Barcelona, Spain.
- 10-13 June.** Brussels. *European Society of Regional Anaesthesia (UK) Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 12-19 June.** The Hague. *10th World Congress of Anaesthesiology*.  
Information: Dr Harm Lip, Nilantweg, 99, 8041 AR Zwolle, Netherlands.
- 9-11 September.** Bournemouth. *Linkman and Annual Scientific Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 17-21 October.** New Orleans. *American Society of Anesthesiologists Annual Meeting*.  
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 12-16 December.** New York. *46th Postgraduate Assembly in Anesthesiology*.  
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists, Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

## 1993

- 12-16 February.** Utah. *38th Annual Postgraduate Course in Anesthesiology — 'Anesthesiology: Today and Tomorrow'*.  
Information: Vicky Larson, Department of Anesthesiology, University of Utah School of Medicine, 50 North Medical Drive, Salt Lake City, Utah 84132, USA.
- 29 April-2 March.** North Carolina. *Meeting of the Association of University Anesthetists*.  
Information: Francis M. James III, Department of Anesthesia, Wake Forest University Medical Center, 300 S. Hawthorne Road, Winston-Salem, North Carolina 27103, USA.
- 1-4 September.** Liverpool. *European Course and Congress in Paediatric Anaesthesia*.  
Information: Dr P.D. Booker, Alder Hey Hospital, Liverpool L12 2AP.
- 22-24 September.** Glasgow. *Linkman Conference and Annual Scientific Meeting*. Joint Meeting between the Association of Anaesthetists of Great Britain and Ireland and the Canadian Anaesthetists' Society.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 9-13 October.** Washington DC. *American Society of Anesthesiologists Annual Meeting*.  
Information: Executive Secretary, ASA 515 Busse Highway, Park Ridge, IL 60068, USA.

## 1994

- 7-9 September.** Brighton. *Linkman Conference and Annual Scientific Meeting*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 2-7 October.** Jerusalem. *European Congress of Anaesthesiology*.  
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.

## **Hazard Notice**

### **Cardiac Records Ltd Model CR2006 Defibrillator HC(Hazard)(90)48 and 49**

This machine is unsuitable for synchronised cardioversion. In two instances this defibrillator delivered its energy at an inappropriate part of the cardiac cycle resulting in ventricular fibrillation. The trigger pulse generated by this unit can occur anywhere within a time window of between 20 and 140 msec. It may be used normally for cardioversion in the unsynchronised mode.

Furthermore, a number of CR2006 defibrillators were sold without the integral ECG recorder. These were designated as CR2004 and are also not suitable for synchronised cardioversion.

## **Safety Action Bulletin**

### **Monitoring probes and sensors used in breathing systems, examination before use: risk of aspiration (SAB(90)84)**

The damaged cover of a thermistor probe which was placed in a breathing system assembly became detached and was inhaled. Probes and sensors placed in breathing systems should be physically checked before use to avoid this problem.

### **Graseby Medical Ltd PEAS syringe pump: risk of overinfusion (SAB(90)85)**

Some cases have been reported of PCAS units where interference caused by removing the AC cord or by an electrostatic discharge transmitted through the key has resulted in the infusion being interrupted or a large bolus dose administered. Customers should have received a letter and modification kit, but if not they should contact the manufacturers.

## Erratum

Anaesthesia, 1991, Volume 46, pages 52–56

### Minimum oxygen requirements during anaesthesia with the Triservice anaesthetic apparatus A study of drawover anaesthesia in the young adult

S. Q. M. Tighe, G. A. Turner, S. B. Merrill and R. J. Pethybridge

As a result of a printer's error, by the time this issue was published the top line in the body of the text of Table 3 in the above paper had been erroneously deleted. The correct version of the table appears below.

Table 3. Mean (SEM)  $P_{aO_2}$ ,  $P_{AO_2}$ ,  $P(A-a)O_2$ , and  $F_{IO_2}$ .

O <sub>2</sub> subgroup (litres/minute)	Ventilation group					
	SV			IPPV		
	0	1	4	0	1	4
$P_{aO_2}$ (kPa)						
Baseline	12.2 (0.6)	12.1 (0.7)	11.3 (0.3)	11.5 (0.3)	11.9 (0.6)	12.0 (0.8)
Intra-operative	8.8* (0.7)	18.9 (2.7)	39.0 (5.4)	12.3 (0.7)	18.7 (1.5)	43.1 (6.8)
$P_{AO_2}$ (kPa)						
Baseline	13.2 (0.3)	13.3 (0.4)	13.1 (0.2)	13.0 (0.2)	13.1 (0.2)	13.2 (0.2)
Intra-operative	11.6 (0.5)	29.6 (2.4)	63.4 (6.6)	13.9 (0.2)	25.5 (0.5)	61.5 (3.5)
$P(A-a)O_2$ (kPa)						
Baseline	1.0 (0.5)	1.2 (0.5)	1.8 (0.2)	1.5 (0.3)	1.2 (0.5)	1.2 (0.7)
Intra-operative	2.8 (0.4)	10.6 (1.7)	24.4 (4.9)	1.6 (0.6)	6.8 (1.8)	18.4 (4.9)
$F_{IO_2}$	0.21 (0.00)	0.40 (0.03)	0.76 (0.07)	0.21 (0.00)	0.33† (0.01)	0.72 (0.04)

SV, spontaneous ventilation; IPPV, intermittent positive pressure ventilation.

$n = 6$  for each subgroup.

\* $p < 0.05$ , compared to both baseline and IPPV (log conversion, ANOVA).

† $p < 0.05$ , compared with SV (ANOVA).

THE ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND

APPLICATION FOR MEMBERSHIP

to

The Association of Anaesthetists of Great Britain and Ireland  
9 Bedford Square,  
London WC1B 3RA, England

(This application will be considered by Council at its next meeting. NO MONEY should be sent in the meantime. After acceptance of the application by Council, the Hon. Secretary will notify the applicant of Council's decision and the amount of subscription payable in the current financial year.)

.....19

To the Honorary Secretary,  
Association of Anaesthetists of Great Britain and Ireland

I (full name) ..... offer my name as a candidate

BLOCK LETTERS

for (a) ORDINARY MEMBERSHIP (b) OVERSEAS MEMBERSHIP (c) ASSOCIATE MEMBERSHIP (d) JUNIOR MEMBERSHIP of the Association (please delete categories not applicable)\*

I hold the following appointment(s) as an anaesthetist (please state grade if in the National Health Service, and indicate the approximate proportion of time spent in the practice of anaesthesia):

Qualifications (with dates).....

(Signed).....

Address.....

.....Hospital Tel. No:.....

.....Home Tel. No:.....

We, being Members† of the Association, nominate as a candidate for ORDINARY MEMBERSHIP/OVERSEAS/ASSOCIATE/JUNIOR MEMBERSHIP

1. (Signed).....

(NAME IN BLOCK LETTERS)

Address.....

BLOCK LETTERS

2. (Signed).....

(NAME IN BLOCK LETTERS)

Address.....

BLOCK LETTERS

\* See the reverse of this page for details of membership categories.

† One sponsor must be an Ordinary Member of the Association.

**THIS PAGE CONCERNS YOU IF YOU ARE**  
**A MEDICALLY QUALIFIED ANAESTHETIST**  
**IN GREAT BRITAIN AND IRELAND, OVERSEAS OR**  
**IN TRAINING**  
**AND ARE NOT YET A MEMBER OF THE**  
**ASSOCIATION OF ANAESTHETISTS**  
**OF GREAT BRITAIN AND IRELAND**  
**OR IF YOU ARE A MEMBER OF ANOTHER**  
**PROFESSION WHO PRACTISES IN ANAESTHESIA**  
**AND YOU WISH TO APPLY FOR ASSOCIATE MEMBERSHIP**

**Ordinary Membership** (*United Kingdom and Ireland only*)

Voting membership of the Association (the only effective body representing anaesthetists' interests in non-academic matters).

The right of attendance at the Annual General and Scientific Meetings and all other meetings of the Association of Anaesthetists.

**Overseas and Associate Membership**

The right of attendance at the Annual Scientific Meeting and other meetings of the Association.

**Junior Membership** (*United Kingdom and Ireland only*)

Voting membership of the Association.

Representation on the Council of the Association. The Chairman and Secretary of the Junior Group sit on Council as voting members.

Attendance at the Annual Scientific Meeting of the Association and at the special Annual Scientific Meeting of the Junior Anaesthetists' Group, at which Junior Members of the Association are given preference.

**Membership Fees** (inclusive of the journal *Anaesthesia*)

*Ordinary Members:* Subscription **£90** (Husband and wife **£64** each)

*Overseas and Associate Members:* Subscription **£90**

*Junior Members:* Subscription **£50** (**£64** after 3 years if still in training)

*Retired Members:* who receive *Anaesthesia* **£15** (there is no subscription for those who do not).

**How to apply for membership**

Fill in the application form on the other side of this page.

Have it countersigned by two members of the Association, one of whom must be an Ordinary Member.

Tear out the page and send it to the address indicated overleaf.

**Do not send money with your application**



# Baillière's International Practice and Research CLINICAL ANAESTHESIOLOGY

## Editorial Board

A.R. Aitkenhead (U.K.), M.J. Cousins (Australia), J.F. Crul (The Netherlands), J.G. Jones (U.K.),  
J.F. Nunn (U.K.), L. Strunin (Canada), D. Thomson (Switzerland), H. Van Aken (Belgium)

*Baillière's Clinical Anaesthesiology* is the essential reference for the busy clinician. Each issue is devoted to a separate topic and provides comprehensive coverage from experts in the field. Containing up-to-the-moment reviews, this international reference provides an invaluable source of current clinical opinion, procedures and techniques in anaesthesiology, supported by relevant research data.

## THE 1991 ISSUES

### No. 1 INFECTION AND THE ANAESTHETIST

Guest Editors:  
C.P. Stoutenbeck (Holland)  
& H.K.F. Van Saene (UK)

### No. 2 INTRAVENOUS ANAESTHESIOLOGY - WHAT IS NEW?

Guest Editors: J.W. Dundee (N.Ireland)  
& J.W. Sear (UK)

### No. 3 KINETICS OF ANAESTHETIC DRUGS IN CLINICAL ANAESTHESIOLOGY

Guest Editor: P.F. White (USA)

## PREVIOUS ISSUE TITLES TO COMPLETE YOUR REFERENCE

### Volume 4, 1990

No. 1  
AUTOMATED ANAESTHETIC RECORDS  
Guest Editor: G. Kenny (U.K.)

No. 2  
INTENSIVE CARE:  
DEVELOPMENTS AND CONTROVERSIES  
Guest Editor: G. Dobb (Australia)

No. 3  
ANAESTHESIA FOR DAY CARE SURGERY  
Guest Editor: T.E.J. Healy (U.K.)

### Volume 3, 1989

No. 1  
ANAESTHESIA FOR THE COMPROMISED HEART  
Guest Editor: P. Foex (U.K.)

No. 2  
METABOLIC RESPONSE TO SURGERY  
Guest Editor: D.R. Bevan (Canada)

No. 3  
DEPTH OF ANAESTHESIA  
Guest Editor: J.G. Jones (U.K.)

## ACKNOWLEDGED BY THE MEDICAL PRESS...

"A plethora of clinically applicable facts and therapy" ... "An excellent source of reference for all clinicians" ... "Up to date" ... "Well written" ... "Practical" ... "Well produced and illustrated" ... "Readable" ... "Well balanced" ... "Excellent source reference" ... "Concise" ... "Comprehensive references" ... "Excellent value" ... "Cost effective" ...

Publication: Volume 5 1991 (3 issues)

Subscription Rate: £41.00 (U.K. and Eire)/£47.00 (All other areas). Single Issues: £27.50 (All areas)



Published by Baillière Tindall  
Harcourt Brace Jovanovich, Publishers



## ORDER FORM: Baillière's Clinical Anaesthesiology

Please return to: Journals Marketing Dept., Harcourt Brace Jovanovich Ltd.,  
24-28 Oval Road, London NW1 7DX, U.K. Tel: 071-267-4466

ISSN: 0950-3501

Publication: Volume 5, 1991 (3 issues)

Prepayment is required

☐ Please enter my subscription for Volume 5, 1991

Back volumes and issues are available  
at current year prices.

☐ Please send me \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

### PRICES:

Annual Subscription: £41.00 (U.K. and Eire)/£47.00 (All other areas).

Single Issues: £27.50 (All areas)

☐ I enclose a cheque for £.....  
(made payable to Harcourt Brace Jovanovich Ltd)

☐ I wish to pay by credit card Amount £.....  
Diners Club / American Express / Visa (Barclaycard) /  
MasterCard (please delete as appropriate)

Card No. \_\_\_\_\_

MasterCard users should add the numbers appearing above their name

Expiry Date \_\_\_\_\_

Signature \_\_\_\_\_

(Your signature is essential, especially when ordering by credit card)

Name \_\_\_\_\_

Address \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_ Date \_\_\_\_\_ JMD/0291/

# Contents: Anaesthesia, vol. 46, no. 4, April 1991

## EDITORIAL

### Ethics in publishing

*G. Smith, R. Miller, J. Saidman and M. Morgan*

255

## ORIGINAL ARTICLES

Double-blind comparison of epidural diamorphine and intramuscular morphine after elective Caesarean section, with computerised analysis of continuous pulse oximetry

*J.D. Stevens, P. Braithwaite, C.F. Corke, T.H. Madej and R.G. Wheatley*

256

Pulse oximeter probes. A comparison between finger, nose, ear and forehead probes under conditions of poor perfusion

*D.G. Clayton, R.K. Webb, A.C. Ralston, D. Duthie and W.B. Runciman*

260

The haemodynamic effects of bronchoscopy. Comparison of propofol and thiopentone with and without alfentanil pretreatment

*A.J. Hill, R.O. Feneck, S.M. Underwood, M.E. Davis, A. Marsh and L. Bromley*

266

The pharmacodynamics of alcuronium in the elderly

*A.P. Kent and J.M. Hunter*

271

Comparison of lumbar plexus block versus conventional opioid analgesia after total knee replacement

*M.G. Serpell, F.A. Millar and M.F. Thomson*

275

Addition of fentanyl to prilocaine for intravenous regional anaesthesia

*P. Armstrong, I. Power and J.A.W. Wildsmith*

278

## CASE REPORTS

Vasovagal asystole during spinal anaesthesia

*I. McConachie*

281

An unusual cause of obstructive emphysema

*R.J. Buist*

283

Milrinone, a bridge to heart transplantation

*D.M. Watson, K.M. Sherry and G.A. Weston*

285

Massive acute arsenic poisoning

*D.M. Jolliffe, A.J. Budd and D.J. Gwilt*

288

## APPARATUS

Potential errors in pulse oximetry. III: Effects of interference, dyes, dyshaemoglobins and other pigments

*A.C. Ralston, R.K. Webb and W.B. Runciman*

291

Assessment of a hygroscopic heat and moisture exchanger for paediatric use

*K.A. Wilkinson, A. Cranston, D.J. Hatch and M.E. Fletcher*

296

## HISTORICAL

Francis Percival de Caux (1892-1965). An anaesthetist at odds with social convention and the law

*D.J. Wilkinson*

300

## FORUM

Intubation guide marks for correct tube placement. A clinical study

*S. Mehta*

306

A comparison of lignocaine with prilocaine in axillary brachial plexus anaesthesia

*E.P. McCoy, and C.M. Wilson*

309

Intubation with propofol augmented with intravenous lignocaine

*D. Mulholland and R.J.T. Carlisle*

312

## CORRESPONDENCE

314

## BOOK REVIEWS

332

## OBITUARIES

335

## INTERNATIONAL CONGRESS CALENDAR

335

## HAZARD NOTICE

337

## SAFETY ACTION BULLETIN

337

## ERRATUM

338



12<sup>11</sup>/<sub>92</sub>

# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 46 Number 5 May 1991



The Association of Anaesthetists of Great Britain and Ireland  
*Patron:* HRH The Princess Margaret, Countess of Snowdon

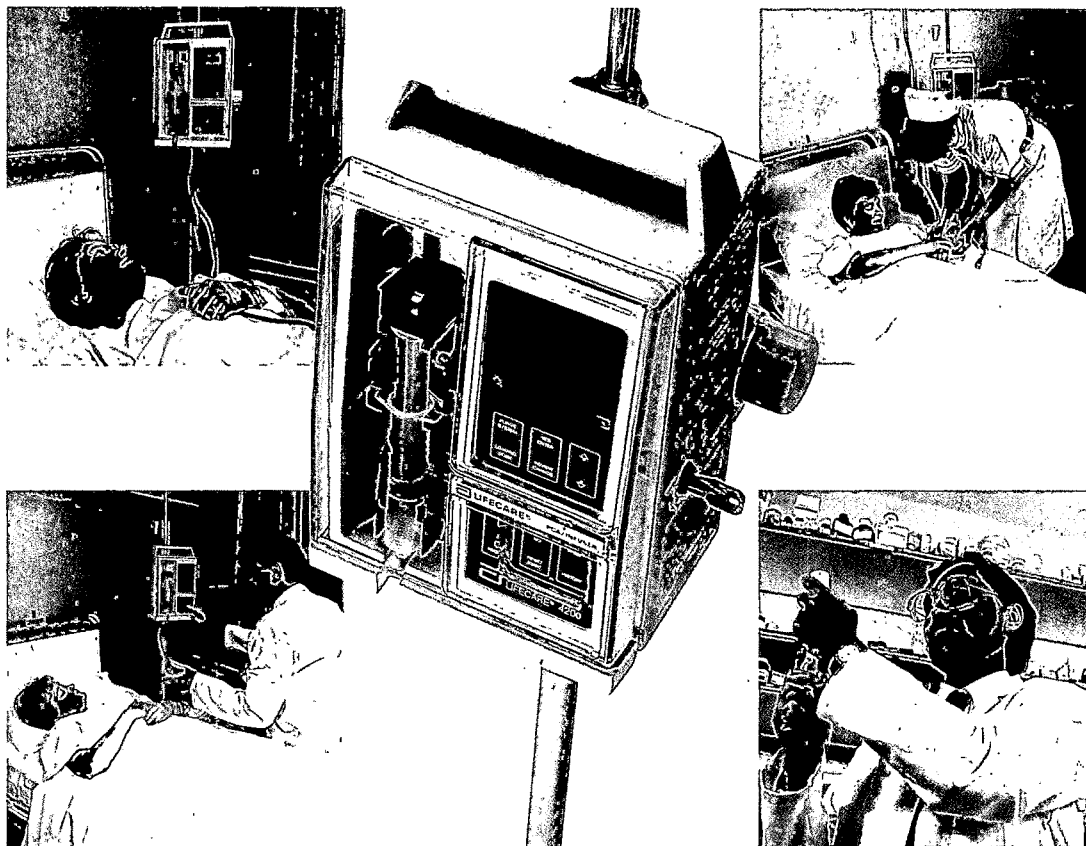


Published for the Association by  
Academic Press Grune & Stratton  
London San Diego New York Boston  
Sydney Tokyo Toronto



ISSN 0003-2409

# ABBOTT PATIENT CONTROLLED ANALGESIA



## Allowing the patient membership of the pain management team...

- + Allows the patient to control their pain.
- + Allows the nurse time to manage the patient.
- + Allows the doctor to establish the dose.
- + Allows the pharmacist to monitor the drug.

If you would like to offer extra membership to your pain management team, please ask for more information from:

**ABBOTT**

**PCAF**

ABBOTT LABORATORIES LIMITED  
Queenborough, Kent ME11 5EL  
Tel: (0795) 580099 Fax: (0795) 580404

# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

---

Editor M. MORGAN

Assistant Editors R. GREENBAUM, A. R. AITKENHEAD, D. E. F. NEWTON, R. A. MASON, M. HARMER

## *Editorial Board*

Chairman The President of the Association P. J. F. BASKETT

Members A. M. ROLLIN, C. A. B. MCLAREN and a representative of the Junior Anaesthetists' Group

## *Ex officio*

The Honorary Treasurer of the Association W. R. MACRAE

The Honorary Secretary of the Association J. E. CHARLTON

The Immediate Past Honorary Secretary of the Association W. L. M. BAIRD

---

DIANE WINTERBURN, JANET SPRINGER, Reference librarians

---

*Address for literary contributions* Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK [telephone 081-740 3290].

Neither the Editor nor the Association of Anaesthetists of Great Britain and Ireland is in any way responsible for the statements made or the views expressed by contributors.

Investigations performed on man must conform to appropriate ethical standards including voluntary, informed consent and acceptance by an ethics committee.

A written statement of this acceptance may be requested by the Editor.

---

ANAESTHESIA: ISSN 0003-2409, Volume 46 1991, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. All advertising enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293). Annual subscription price including postage: £132 UK and US \$244 overseas. Personal subscription rate including postage: \$99. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel. 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

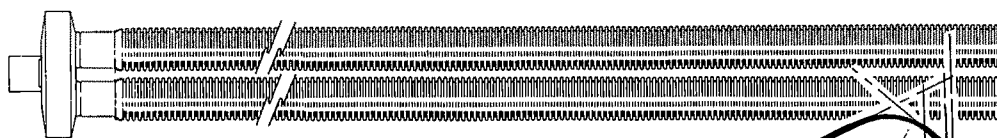
Printed in UK on acid-free paper.

*Anaesthesia* is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

© 1991 The Association of Anaesthetists of Great Britain and Ireland. The appearance of the code at the bottom of the first page of a paper in this journal indicates the copyright owner's consent that copies of the paper may be made for personal or internal use, or for the personal or internal use of specific clients in the USA. This consent is given on the condition, within the USA, that the copier pay the stated per-copy fee through the Copyright Clearance Center Inc., 27 Congress Street, Salem, MA 01970, USA for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to other kinds of copying, such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, for resale or for copying or distributing copies outside the USA. This journal is printed and bound in Great Britain by BPCC Wheatons Ltd, Exeter.

# The safer alternative

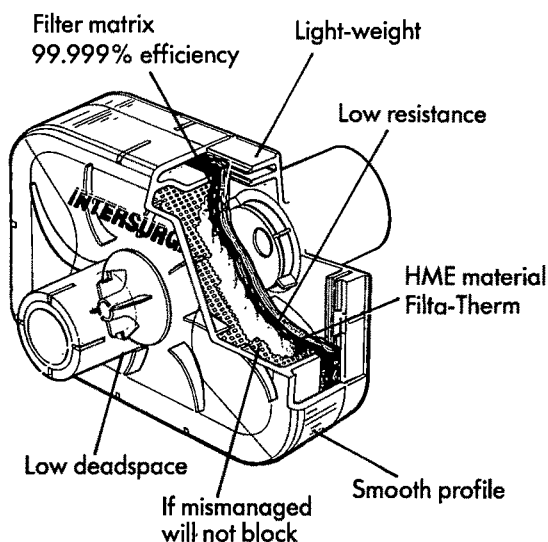
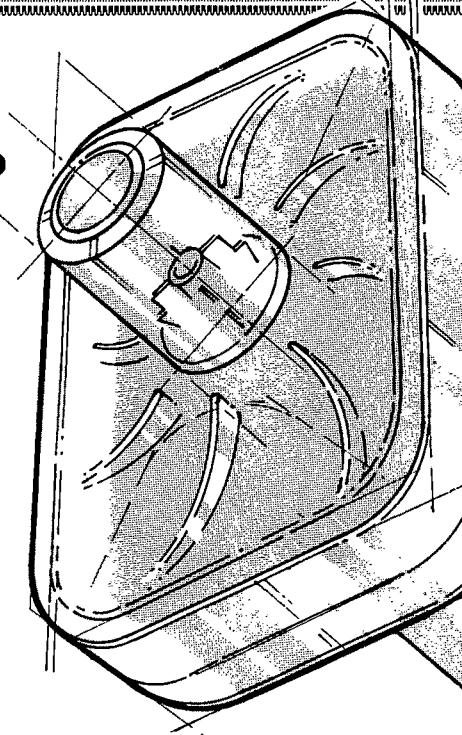
## Filtration Technology at its best



**A range of unique Breathing Filters/HME's to resolve all your filtration and humidification problems**

The Filta-Therm – providing both Heat and Moisture Exchange and Bacterial/Viral Filtration 99.999% – ideal for **patient protection and humidification.**

The Filta-Guard – providing Bacterial/Viral Filtration 99.999% – ideal for **equipment protection – no wastage.**



**Choose the ideal product for the clinical situation**

**Choose a safe product – choose Intersurgical.**

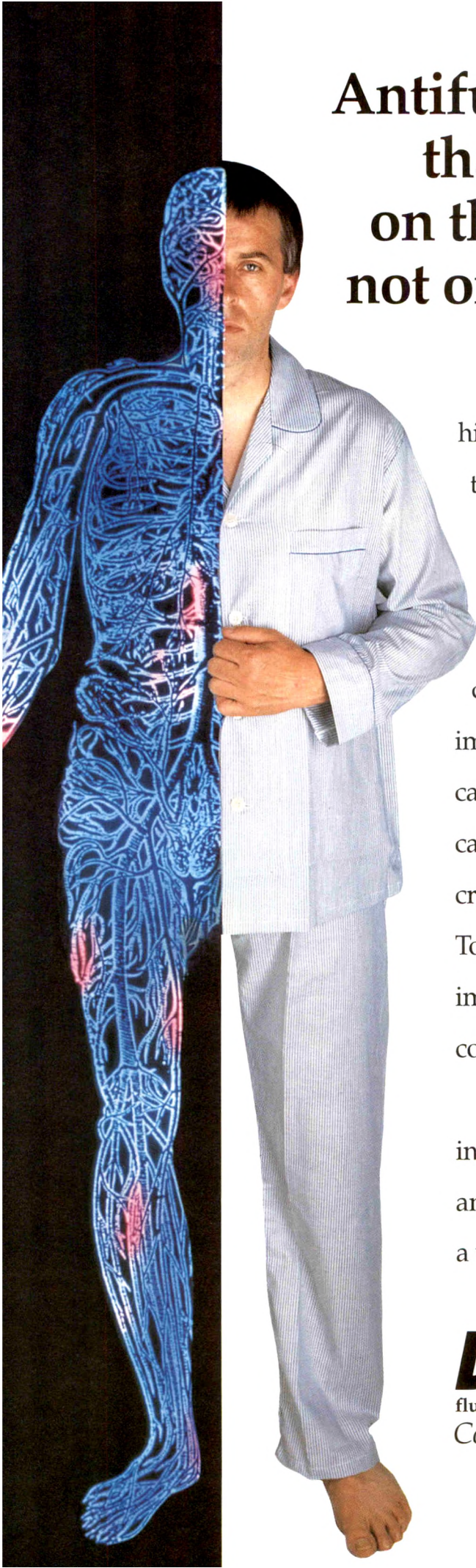
**INTERSURGICAL**  
COMPLETE RESPIRATORY SYSTEMS

Crane House, Gould Road,  
Twickenham, Middlesex TW2 6RS.  
Tel: 081-755 1477, Fax: 081-755 2177

I am interested in further details of Filta-Guard ☐ Filta-Therm ☐  
Intersurgical, Crane House, Gould Road, Twickenham, Middlesex TW2 6RS.

Name	Position	Dept.
Hospital	Hospital Address	
Equipment used		





# Antifungal therapy that's severe on the infection, not on the patient.

Diflucan Oral/I.V. is a highly effective, well tolerated therapy with a convenient, once a day dosage and administration.

Overall clinical responses in deep-seated fungal infections are impressive: 87% in systemic candidiasis<sup>1</sup>, 96% in mucosal candidiasis<sup>2</sup> and 62% in cryptococcal meningitis.<sup>3</sup> Tolerability is similarly impressive with adverse effects comparable to placebo.<sup>4</sup>

Diflucan Oral/I.V. Treating invasive fungal infection with an iron fist and patients with a velvet glove.

**Diflucan** ORAL/I.V.  
fluconazole \*Trade Mark

*Counters invasive fungal infection*



**The first true choice in sophisticated  
data management.**



**The Modulus CD Anaesthesia System**

**Ergonomically organised. Medu**

## Editorial

### Ropivacaine

In 1957 Ekenstam *et al.*<sup>1</sup> described the synthesis and physicochemical properties of a series of N-alkyl piperidine carboxylic acid amides, or, as they are sometimes described, N-substituted piperidyl xylidines. This series includes mepivacaine, bupivacaine and the more recently investigated propyl derivative, the S isomer of which is now called ropivacaine.

These piperidyl xylidines derivatives, together with prilocaine and etidocaine, are what is known as chiral drugs, that is they all possess an asymmetric carbon atom and exist as two stereoisomers, once termed the L and D forms (*laevo* and *dextro*) and now S and R (*sinister* and *rectus*). (I for one would prefer to call them L and R, implying left- and right-handedness, and eschew this obsession with the classics.) It has been shown that the L or S forms of prilocaine,<sup>2</sup> bupivacaine<sup>3</sup> and mepivacaine<sup>4</sup> are longer acting than the D or R forms, and this can generally be attributed to the vasoconstrictor activity vested in one isomer, although a difference in pharmacological activity has also been postulated.<sup>4</sup>

Large doses of bupivacaine injected intravenously, either accidentally or deliberately in experimental animals and in Bier's blocks, are potentially cardiotoxic. Because of this, an agent with a wider margin of safety has been sought and the S isomer of N-propyl piperidyl xylidine selected as a candidate for this role. As would be predicted from its position in the homologous series, ropivacaine is less lipid-soluble than bupivacaine, and the pKa values of the two agents are approximately equal at about 8.1.<sup>5</sup> Although modern racemic local anaesthetics tend to be vasodilator at high concentrations and vasoconstrictor at lower,<sup>3</sup> ropivacaine appears to be vasoconstrictor over a wide range of concentrations.<sup>6</sup> Preliminary trials in animals<sup>7</sup> showed it to be longer acting than the same concentrations of bupivacaine when used for infiltration and about equal for sciatic and brachial plexus block, but shorter in duration via the epidural route. *In vitro* testing suggests that the selectivity of ropivacaine for sensory fibres may be at least as good as that of bupivacaine in rat<sup>8</sup> and rabbit,<sup>9</sup> while *in vivo* potency and duration of motor blockade with ropivacaine would appear to be slightly less than that with bupivacaine in the rat sciatic nerve and dog epidural space.<sup>10</sup> Its half-life in dog<sup>11</sup> and man<sup>12</sup> appears to be shorter than that of bupivacaine which would make it potentially safer for repeated use.

Clinically, ropivacaine has been used by a number of routes and over a range of concentrations from 0.5 to 1%<sup>13–15</sup> and to produce very effective epidural blockade, by which route it would appear to be only slightly less potent and long acting than bupivacaine.<sup>16</sup>

But what of its potential for cardiotoxicity? Riez *et al.*<sup>17</sup> showed that the intravenous dose of ropivacaine required to prolong the QRS complex in pigs was more than double that of bupivacaine, while Feldman *et al.*<sup>18</sup> showed that in dogs, although the convulsant doses of

ropivacaine and bupivacaine were similar, convulsant plasma concentrations of ropivacaine were actually lower, but the potential for causing death from ventricular fibrillation, although present, was less. In sheep, the intravenous fatal dose was double that of bupivacaine but, unlike lignocaine, both caused ventricular arrhythmias.<sup>19</sup> The convulsant dose of ropivacaine administered epidurally was one third greater than that of bupivacaine, but at this dose ratio the arrhythmogenic potential of the two appeared to be similar.<sup>20</sup> Ropivacaine, given epidurally in this dose ratio to dogs, produced a greater prolongation of the PR interval and QRS complex than did bupivacaine, while heart rate and blood pressure were reduced similarly by the two drugs.<sup>21</sup> In human volunteers Scott *et al.* demonstrated little difference in central nervous and cardiovascular toxic potential between ropivacaine and bupivacaine given intravenously; ropivacaine was about 25% less potent in both respects.<sup>22</sup> Truly toxic doses cannot of course be given to human volunteers deliberately, however, and the question remains whether we more closely resemble pigs, dogs or sheep in our response to these drugs.

In this issue Kerkkamp and Gielen (pages 361–5) report on the haemodynamic changes associated with lumbar epidural administration of bupivacaine and ropivacaine in equal doses of 150 mg with adrenaline. There was a tendency for ropivacaine to produce more tachycardia, less hypotension and less increase in cardiac output, although onset and spread of analgesia were similar with the two agents. While the vasoconstrictor activity of ropivacaine is likely to enhance its efficacy and duration of blockade in the epidural space, making it more effective than would be predicted from its position within the homologous series, systemic concentrations of the drug are unlikely to be sufficient to produce vasoconstriction by a peripheral action. Thus, a difference in the extent of blockade of B fibres in the white rami communicantes is more likely to account for a difference in hypotensive potential between the two agents, while the increased cardiac output with bupivacaine is an expected reflex response to such hypotension.

Use of the single more active enantiomer rather than a racemic mixture of a chiral drug is clearly an advance and may well be the way ahead in many therapeutic spheres.<sup>23</sup> Hitherto single isomers have been expensive to produce, but techniques are advancing rapidly and may well in the future overcome these financial constraints. Although in the UK bupivacaine cardiotoxicity has not been a major problem except with intravenous regional anaesthesia, nevertheless ropivacaine is to be welcomed as a potential means of providing reliable and long lasting regional blockade for surgery. Available evidence suggests its potency is at least 0.75 that of bupivacaine, and although its margin of safety for seizures is similar, its cardiotoxic potential is probably less. Moreover, given its long duration but short

systemic half-life and high protein binding it may be of real value for epidural analgesia in obstetrics.

*St Thomas' Hospital,  
London SE1 7EH*

F. REYNOLDS

### References

1. EKENSTAM B AF, EGNER B, PETTERSSON G. N-alkyl pyrrolidine and N-alkyl piperidine carboxylic acid amides. *Acta Chemica Scandinavica* 1957; **11**: 1183.
2. AKERMAN B, PERSSON H, TEGNER C. Local anaesthetic properties of the optically active isomers of prilocaine (Citanest). *Acta Pharmacologica et toxicologica* 1967; **25**: 233-41.
3. APS C, REYNOLDS F. An intradermal study of the local anaesthetic and vascular effects of the isomers of bupivacaine. *British Journal of Clinical Pharmacology* 1978; **6**: 63-8.
4. FAIRLEY JW, REYNOLDS F. An intradermal study of the local anaesthetic and vascular effects of the isomers of mepivacaine. *British Journal of Anaesthesia* 1981; **53**: 1211-16.
5. ROSENBERG PH, KYTTA J, ALILA A. Absorption of bupivacaine, etidocaine, lignocaine and ropivacaine into N-heptane, rat sciatic nerve and human extradural and subcutaneous fat. *British Journal of Anaesthesia* 1986; **58**: 310-14.
6. KOPACZ DJ, CARPENTER RL, MACKEY DC. Effect of ropivacaine on cutaneous capillary blood flow in pigs. *Anesthesiology* 1989; **71**: 69-74.
7. AKERMAN B, HELLBERG I-B, TROSSVIK C. Primary evaluation of the local anaesthetic properties of the amino amide agent ropivacaine (LEA 103). *Acta Anaesthesiologica Scandinavica* 1988; **32**: 571-8.
8. ROSENBERG PH, HEINONEN E. Differential sensitivity of A and C nerve fibres to long-acting amide local anaesthetics. *British Journal of Anaesthesia* 1983; **55**: 163-7.
9. BADER AM, DATTA S, FLANAGAN H, COVINO BG. Comparison of bupivacaine- and ropivacaine-induced conduction blockade in the isolated rabbit vagus nerve. *Anesthesia and Analgesia* 1989; **68**: 724-7.
10. FELDMAN HS, COVINO BG. Comparative motor-blocking effects of bupivacaine and ropivacaine, a new amino amide local anesthetic in the rat and dog. *Anesthesia and Analgesia* 1988; **67**: 1047-52.
11. ARTHUR GR, FELDMAN HS, COVINO BG. Comparative pharmacokinetics of bupivacaine and ropivacaine, a new amide local anesthetic. *Anesthesia and Analgesia* 1988; **67**: 1053-8.
12. LEE A, FAGAN D, LAMONT M, TUCKER GT, HALLDIN M, SCOTT DB. Disposition kinetics of ropivacaine in humans. *Anesthesia and Analgesia* 1989; **69**: 736-8.
13. KATZ JA, BRIDENBAUGH PO, KNARR DC, HELTON SH, DENSON DD. Pharmacodynamics and pharmacokinetics of epidural ropivacaine in humans. *Anesthesia and Analgesia* 1990; **70**: 16-21.
14. CONCEPCION M, ARTHUR GR, STEELE SM, BADER AM, COVINO BG. A new local anesthetic ropivacaine. Its epidural effects in humans. *Anesthesia and Analgesia* 1990; **70**: 80-5.
15. WHITEHEAD E, ARRIGONI B, BANNISTER J. An open study of ropivacaine in extradural anaesthesia. *British Journal of Anaesthesia* 1990; **64**: 67-71.
16. BROWN DL, CARPENTER RL, THOMPSON GE. Comparison of 0.5% ropivacaine and 0.5% bupivacaine for epidural anesthesia in patients undergoing lower extremity surgery. *Anesthesiology* 1990; **72**: 633-6.
17. REIZ S, HAGGMARK S, JOHANSSON G, NATH S. Cardiotoxicity of ropivacaine — a new amide local anesthetic agent. *Acta Anaesthesiologica Scandinavica* 1989; **33**: 93-8.
18. FELDMAN HS, ARTHUR GR, COVINO BG. Comparative systemic toxicity of convulsant and supraconvulsant doses of intravenous ropivacaine, bupivacaine and lidocaine in the conscious dog. *Anesthesia and Analgesia* 1989; **69**: 794-801.
19. NANCARROW C, RUTTEN AJ, RUNCIMAN WB, MATHER LE, CARAPETIS RJ, MCLEAN CF, HIPKINS SF. Myocardial and cerebral drug concentrations and the mechanisms of death after fatal intravenous doses of lidocaine, bupivacaine and ropivacaine in sheep. *Anesthesia and Analgesia* 1989; **69**: 276-83.
20. RUTTEN AJ, NANCARROW C, MATHER LE, ILSLEY AH, RUNCIMAN WB, UPTON RN. Hemodynamic and central nervous system effects of intravenous bolus doses of lidocaine, bupivacaine and ropivacaine in sheep. *Anesthesia and Analgesia* 1989; **69**: 291-9.
21. FELDMAN HS, ARTHUR GR, COVINO BG. Comparative hemodynamic effects of epidurally administered solutions of ropivacaine and bupivacaine plain and containing epinephrine in the dog. *Anesthesia and Analgesia* 1989; **68**: S84.
22. SCOTT DB, LEE A, FAGAN D, BOWLER GMR, BLOOMFIELD P, LUNDH R. Acute toxicity of ropivacaine compared with that of bupivacaine. *Anesthesia and Analgesia* 1989; **69**: 563-9.
23. EMSLEY J. Split route to a drug revolution. *The Independent*, 18th June 1990.

### Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; **1**: 432-5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

## The neuromuscular blocking effect of Org 9426

A new intermediately-acting steroidal non-depolarising muscle relaxant in man

L. H. D. J. BOOIJ AND H. T. A. KNAPE

### Summary

Org 9426, a new steroidal non-depolarising muscle relaxant, which is stable in solution, was studied in 30 anaesthetised (thiopentone, fentanyl, nitrous oxide) male patients, ASA 1 or 2. A dose-response curve for Org 9426 was constructed and the  $ED_{50}$ , mean (SD), was found to be 0.36 (0.031) mg/kg. The onset of action was 3.8 (1.8) minutes, the clinical duration 17.4 (3.2) minutes, the total duration of action 31.9 (6.2) minutes and the recovery time 9.96 (3.2) minutes. No signs of histamine release or cardiovascular effects were observed. Org 9426 thus has a faster onset of action than vecuronium bromide or atracurium dibesylate.

### Key words

Neuromuscular relaxants; Org 9426.

The introduction of vecuronium bromide and atracurium dibesylate improved considerably clinical flexibility in the administration of muscle relaxants. The durations of action of these compounds are more suitable to the duration of action required for the majority of operations than that of any other clinically used non-depolarising muscle relaxant.

However, vecuronium bromide is not stable in solution and atracurium dibesylate is known to release histamine even at clinically used doses. In animal experiments Org 9426, 1-(17 $\beta$ -acetoxy-3 $\alpha$ -hydroxy-2 $\beta$  morpholino-5 $\alpha$ -androstane-16 $\beta$ -yl)-1-allylpyrrolidinium bromide, has been demonstrated to be a non-depolarising muscle relaxant with a time course of action comparable to that of vecuronium bromide,<sup>1,2</sup> but contrary to vecuronium bromide Org 9426 is stable in solution.

Animal studies proved Org 9426 to be free from cardiovascular effects,<sup>3</sup> and in acute toxicological studies in two species, toxic effects on organs and changes in biochemical variables could not be demonstrated, even at high doses up to 10 times the  $ED_{50}$  in the relevant species (data on file with Organon Teknika). We report here the neuromuscular blocking effect of Org 9426 in anaesthetised man.

### Methods and materials

Written informed consent was obtained from 30 male patients, aged 21 to 50 years, to participate in the study which was approved by the Hospital Ethics Committee. All the patients had normal renal and hepatic function and were not receiving medication that might interfere with neuromuscular transmission.

The patients were premedicated with diazepam 0.1 mg/kg orally, 45–60 minutes before induction of anaesthesia. An intravenous infusion was started and anaesthesia was induced with thiopentone 4 mg/kg and fentanyl 0.003–0.005 mg/kg. Maintenance was with nitrous oxide 67% in oxygen delivered by mask and fentanyl 0.05 mg bolus when needed, as judged by clinical signs. The electrocardiogram and noninvasive blood pressure (Finapres, Ohmeda) were recorded continuously. Ventilation of the lungs was controlled to keep the end-tidal carbon dioxide partial pressure at 4 kPa. The temperature of the patients was between 36.5 and 37.5°C.

The right ulnar nerve was stimulated near the wrist *via* surface electrodes with square wave supramaximal stimuli at a rate of 0.1 Hz and 0.2 mseconds duration, delivered by a Grass S44 stimulator. The resulting contractions of the *adductor pollicis* muscle were quantitated by a force displacement transducer and recorded on a polygraph. When the elicited twitch contractions were stable for 5 minutes, one dose of Org 9426 was administered to each patient *via* a rapid infusion. A dose-response curve was constructed from the depressions of the force of contraction (by fitting a bi-exponential curve) and the  $ED_{50}$  and the  $ED_{90}$  (doses resulting in 50 and 90% depression) calculated.

When the depression of twitch contraction was more than 80%, the onset of action (time from end of injection to maximal effect), the clinical duration (time from end of injection to 25% recovery), the total duration (time from end of injection to 90% recovery), and the recovery time (time from 25% to 75% recovery) were estimated. The values are given as mean (SD). The same was done in the

L.H.D.J. Booiij, MD, PhD, Professor and Chairman, H.T.A. Knape, MD, PhD, Consultant, Institute for Anaesthesiology, University of Nijmegen, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands.

Accepted 30 June 1990.

group of patients where complete paralysis was obtained; mean and standard deviation for all variables were calculated. In addition patients were observed for gross changes in heart rate and blood pressure as well as for irregularities in the ECG.

### Results

Twenty-eight patients were included in the study and received Org 9426 in doses of 0.11 to 2.5 mg/kg (Table 1). Two patients were excluded from the study because, later on, it appeared that they did not fulfil the inclusion criteria.

The dose-response curve (Fig. 1) reveals an  $ED_{50}$  of 0.22 (SD 0.016) mg/kg and an  $ED_{90}$  of 0.36 (0.031) mg/kg. Blockade of more than 80% and less than 100% was obtained in seven patients (patients 9, 10, 17, 22, 23, 24, 26 in Table 1). The mean administered dose for these seven patients was 0.04 (0.11) mg/kg leading to a blockade of 93.9 (3.6)%, with an onset of action of 3.8 (1.8) minutes, a clinical duration of 18.8 (4.5) minutes, a total duration of 32.1 (6.3) minutes, and a recovery time of 9.96 (3.2) minutes (Table 2).

Complete paralysis was obtained in another seven patients (numbers 11, 16, 18, 19, 20, 21, 25 in Table 1) with a mean dose of 0.38 (0.07) mg/kg, an onset of 1.7 (0.77) minutes, a clinical duration of 18.9 (5.53) minutes, a total duration of 33.9 (12.81) minutes, and a recovery time of 7.7 (3.38) minutes. The patients who received very large doses (numbers 27 and 28) were not included in this last analysis.

There were no observed effects on the ECG, heart rate or blood pressure that could be attributed to the administration of Org 9426, nor were there any effects in the two patients who received three or six times the  $ED_{90}$  (numbers 27 and 28). No clinical signs of histamine release were found.

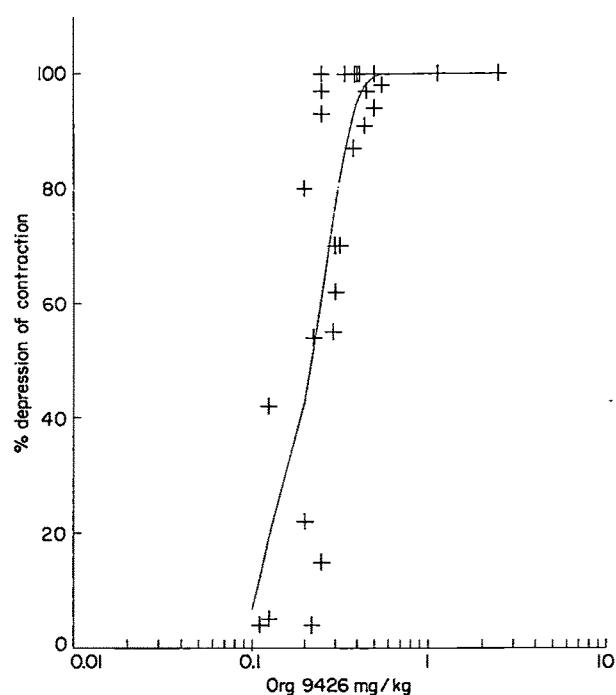


Fig. 1. Dose response curve for Org 9426. Each + represents a measured point (at 0.4 mg/kg two points are overlapping for 100% block).

### Discussion

Org 9426 is a non-depolarising muscle relaxant which is stable in solution. There is some variability in the sensitivity for this relaxant amongst individual patients as with all muscle relaxants. This variability has to be regarded as a

Table 1. Neuromuscular blocking effect of Org 9426 in individual patients.

n	Dose (mg/kg)	Block (%)	Onset (minutes)	Duration (minutes to 25%)	Duration (minutes to 90%)	Recovery rate (minutes)
1	0.11	4	—	—	—	—
2	0.125	5	—	—	—	—
3	0.125	42	—	—	—	—
4	0.2	22	—	—	—	—
5	0.2	80	—	—	—	—
6	0.22	4	—	—	—	—
7	0.225	54	—	—	—	—
8	0.25	15	—	—	—	—
9	0.25	93	4.3	14.0	25.0	8.0
10	0.25	97	1.0	19.3	24.3	5.4
11	0.25	100	1.0	18.95	35.3	8.9
12	0.29	55	—	—	—	—
13	0.3	62	—	—	—	—
14	0.3	70	—	—	—	—
15	0.32	70	—	—	—	—
16	0.34	100	1.3	16.0	37.0	13.2
17	0.38	87	3.0	16.0	26.0	12.4
18	0.39	100	2.5	14.0	18.7	3.6
19	0.4	100	2.0	18.6	27.4	6.6
20	0.4	100	0.75	22.4	56.0	5.5
21	0.41	100	2.0	12.1	18.0	4.4
22	0.44	91	3.6	18.0	39.0	15.3
23	0.45	97	7.2	23.0	34.0	9.0
24	0.5	94	3.5	14.0	37.3	9.3
25	0.5	100	2.6	30.0	45.0	11.5
26	0.55	98	2.4	27.0	39.0	9.8
27	1.14	100	1.5	103.0	150.0	42.6
28	2.5	100	0.7	—	—	—



Table 2. Time course of action of Org 9426 as compared to other non-depolarising muscle relaxants.  
\* = reference where data are obtained.

Compound	ED <sub>90</sub>	Onset (minutes)	Duration (minutes to 25%)	Duration (minutes to 90%)	Recovery rate (minutes)	*
Org 9426	0.40	3.8 (1.8)	17.4 (3.2)	31.9 (6.2)	9.96 (3.2)	
Vecuronium	0.05	4.5 (0.2)	11.6 (1.2)	24.9 (2.4)	9.7 (0.8)	4
Pancuronium	0.06	4.9 (0.7)	34.4 (5.8)	73.2 (11.4)	31.9 (4.5)	4
Pipecurium	0.05	6.7 (0.4)	29.0 (2.0)	—	31.0 (3.1)	8
Alcuronium	0.14	5.9 (0.4)	29.3 (1.6)	62.6 (7.5)	29.0 (2.3)	4
Tubocurarine	0.34	9.9 (1.1)	33.8 (4.3)	96.4 (16.1)	59.3 (8.1)	4
Mivacurium	0.10	3.8 (0.5)	14.2 (1.5)	24.5 (1.6)	7.0 (0.5)	5
Atracurium	0.19	6.7 (0.4)	17.1 (1.4)	32.0 (1.6)	12.0 (0.5)	6
Doxacurium	0.03	10.2 (1.3)	84.3 (12.4)	128.4 (26.4)	10.6 (8.4)	7

normal interindividual variation occurring in the effect of most drugs and in most biological functions.

The calculated ED<sub>90</sub> of 0.36 mg/kg is lower than the mean dose of 0.40 mg/kg with which an average block of 94% was reached in seven patients. Another seven patients reached complete paralysis with 0.38 mg/kg.

The onset of blockade is shortened by administration of a higher dose. The durations of action of respectively 17.4 and 18.9 minutes for clinical duration and 31.9 and 33.9 minutes for the total duration, seem to be less prolonged with deeper blockade.

This decrease in onset may be explained from an initial rapid decrease in force of contraction, followed by a secondary slower decrease observed with the use of this compound. In the case of 94% depression of force of contraction this will delay the onset, as defined, whereas in the case of 100% depression this last phase cannot be observed. This biphasic onset does not significantly affect the duration of action (see for explanation Fig. 2).

The present study is not a real study on the conditions for tracheal intubation, but we could easily intubate the patients who ultimately developed 90% or deeper blockade, at about 1.5 minutes after administration of Org 9426. Figure 2 shows that in most patients an approximately 70 to 80% blockade existed. This rapid initial decrease enables early intubation, which is an advantage over the use of vecuronium bromide or atracurium dibesyl-

late, where the depression of the twitch contraction is more gradual. Proof of this, however, has to be obtained from more extensive studies on intubation conditions. Org 9426 was free in the present study from cardiovascular side effects and histamine release, but further, more detailed studies on these aspects have to be performed.

Org 9426 seems to be comparable to vecuronium bromide,<sup>4</sup> mivacurium chloride<sup>5</sup> and atracurium dibesylate<sup>6</sup> when comparisons are made with other non-depolarising relaxants. The duration of action is shorter than that of doxacurium chloride,<sup>7</sup> pipecurium bromide,<sup>8</sup> pancuronium bromide, alcuronium, or tubocurarine.<sup>4</sup> Org 9426 is a promising compound that deserves further clinical investigation.

## References

1. BOOIJ LHDJ, MARSHALL IG, CRUL JF, MUIR AW. Pharmacology of four steroidal muscle relaxants. Abstracts of the 9th World Congress of Anesthesiologists, Washington 1988, Vol. II: A0533.
2. MARSHALL RJ, MUIR AW, BOOIJ LHDJ, CRUL JF, MARSHALL IG. The cardiovascular effects of four new non-depolarizing neuromuscular blocking drugs in rats, cats, dogs, pigs and monkeys. Abstract of the 9th World Congress of Anesthesiologists, Washington 1988, Vol. II: A0534.
3. BOOIJ LHDJ, CRUL JF, VAN DER POL FF. Cardiovascular and neuromuscular blocking effects of four new muscle relaxants in anaesthetized beagle dogs. *European Journal of Anaesthesiology* 1989; **9**: 70.
4. KRIEG N, CRUL JF, BOOIJ LHDJ. Relative potency of Org NC45, pancuronium, alcuronium and tubocurarine in anaesthetized man. *British Journal of Anaesthesia* 1980; **52**: 783-7.
5. SAVARESE JJ, ALI HH, BASTA SJ, EMBREE PB, SCOTT RPF, SUNDER N, WEAKLY JN, WASTILA WB, EL-SAYAD HA. The clinical neuromuscular pharmacology of mivacurium chloride (BW-B1090U). *Anesthesiology* 1988; **68**: 723-32.
6. ROBERTSON EN, BOOIJ LHDJ, FRAGEN RJ, CRUL JF. Clinical comparison of atracurium and vecuronium (Org NC45). *British Journal of Anaesthesia* 1983; **55**: 125-9.
7. BASTA SJ, SAVARESE JJ, ALI HH, EMBREE PB, SCHWARTZ AF, RUDD GD, WASTILA WB. Clinical pharmacology of doxacurium chloride. A new long-acting nondepolarizing muscle relaxant. 1988; **69**: 478-86.
8. WIERDA JMKH, RICHARDSON FJ, AGOSTON S. Dose-response relation and time course of action of pipecurium bromide in humans anesthetized with nitrous oxide and isoflurane, halothane, or droperidol and fentanyl. *Anesthesia and Analgesia* 1989; **68**: 208-13.

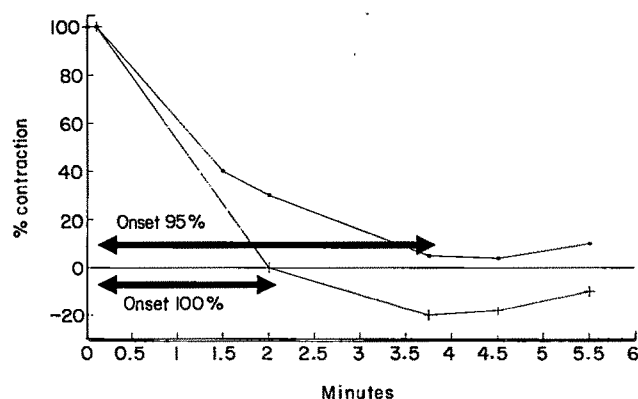


Fig. 2. Onset of action for the ED<sub>55</sub> and for the ED<sub>100</sub> dose of Org 9426.

## The effect of prophylactic fentanyl on shivering in elective Caesarean section under epidural analgesia

W. H. D. LIU AND M. C. LUXTON

### Summary

*The aims of this randomised double-blind study were to investigate whether 25 µg of fentanyl administered prophylactically by the epidural route would influence the incidence of shivering in parturients who underwent elective Caesarean section under epidural analgesia and whether it would affect the axillary and calf temperatures. There was a 50% reduction ( $p < 0.05$ ) in the overall incidence of shivering in patients who received fentanyl and there was some evidence to suggest that low-dose epidural fentanyl might reduce shivering by an influence on thermoregulation.*

### Key words

*Anaesthetic technique, regional; epidural.*

*Analgesics, narcotic; fentanyl.*

*Complications; shivering.*

*Monitoring; temperature.*

Shivering has been reported to occur in 20% of patients in labour<sup>1</sup> and up to 61% of patients who undergo lower segment Caesarean section with epidural analgesia.<sup>2</sup> Shivering during Caesarean section may distress patients and in severe cases may interfere with monitoring such as electrocardiography, blood pressure measurement and pulse oximetry.

It has been suggested that epidural fentanyl is useful in reducing shivering during epidural analgesia for pain relief.<sup>3</sup> However, it is not known whether prophylactic epidural fentanyl is effective in reducing shivering during epidural Caesarean section, when larger doses of local anaesthetics are employed and the abdominal cavity is exposed to ambient temperature. It has also been suggested that hypothermia during Caesarean section under epidural anaesthesia is due mostly to redistribution of body heat, rather than exaggerated heat loss to the environment.<sup>3</sup> We therefore investigated whether prophylactic epidural fentanyl 25 µg reduces the incidence of shivering, and monitored its effect on axillary and calf temperatures in patients who underwent elective Caesarean section under epidural analgesia.

### Methods

This was a randomised, double-blind, prospective study in 41 healthy (ASA grade 1 or 2) parturients, who were

between 18 and 35 years of age, gravidity of three or less, and scheduled for elective Caesarean section under epidural analgesia because of previous Caesarean section or cephalopelvic disproportion. The parturients were assigned into two groups by block randomisation. Group F received epidural fentanyl in addition to a local anaesthetic agent; group P received preservative-free saline 0.9% as placebo with the local anaesthetic. The study was approved by the Hospital Ethics Committee and written informed consent was obtained from all parturients.

Parturients were not studied if they were receiving medications which might influence thermoregulation, if the intra-operative blood loss was more than one litre or if they were shivering before the commencement of the epidural. All parturients were premedicated with ranitidine 150 mg at 2200 hours on the evening before and at 0600 hours on the day of the operation. All operations were performed in the morning.

Baseline measurements were made of blood pressure, heart rate, maternal axillary and midcalf cutaneous temperatures (measured by a two channel digital thermometer EXACON MC8700 equipped with automatic calibration) on arrival in the anaesthetic room. The axillary temperature was measured by an electrode which was secured by adhesive tapes and placed at the apex of the right axilla of each patient with the right arm adducted across the chest. The midcalf temperature was measured by

W.H.D. Liu, MB, BCh, FCAnaes, Research Fellow, M.C. Luxton, BM, BCh, FCAnaes, Consultant, University Department of Anaesthesia, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH.

Accepted 23 August 1990.

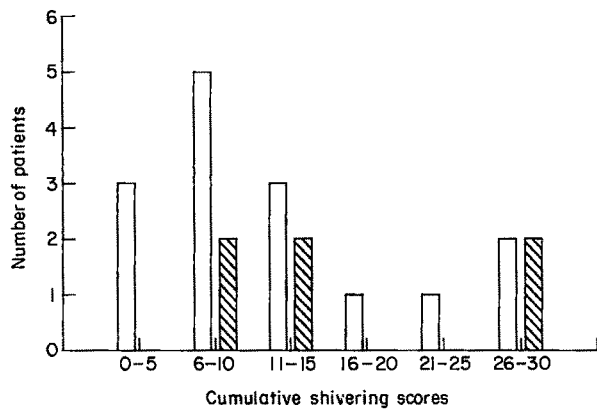


Fig. 1. Frequency distribution of cumulative shivering scores (▨, fentanyl; □, placebo).

a second electrode which was secured by adhesive tapes and placed on the outer aspect of the right calf of all patients. The ambient room temperature (measured by a wall-mounted mercury-in-glass thermometer unit) was recorded. Intravenous access was then established. One litre of Ringer's solution was infused over 30 minutes, followed by 500 ml of gelatin solution. Subsequent fluid requirement alternated between Ringer's and gelatin solution. All intravenous fluids were administered at room temperature. Blood pressure was maintained at above 100 mmHg systolic with fluids and boluses of ephedrine 3 mg if necessary.

The epidural space was located between the second and the third or the third and fourth lumbar vertebrae, by the loss-of-resistance-to-air technique. The catheter was primed with saline 0.9%. A coded syringe was prepared by the staff on the delivery suite; it contained either fentanyl 25 µg made up to 2 ml with saline 0.9%, or 2 ml of preservative-free saline 0.9% only. This solution was then mixed with 3 ml of bupivacaine 0.5% with 1:200,000 adrenaline. This 5 ml volume was administered as the test dose (time 0). The correct placement of the epidural catheter was confirmed. Then, doses of 7 ml and 10 ml of bupivacaine 0.5% with adrenaline 1:200,000 were administered at 5 and 15 minutes respectively. The level of block was tested with an ethyl chloride spray. An upper level of block which extended to the T<sub>4</sub> dermatome was considered satisfactory. Further increments of 5–10 ml of bupivacaine 0.5% with adrenaline were given if the block had not reached the T<sub>4</sub> dermatome at 30 minutes. All injectates of local anaesthetic were given at room temperature. Analgesic supplements of papaveretum 5 mg were administered intravenously if required during the operation. Parturients who complained of nausea and/or vomiting which necessitated treatment were given metoclopramide 10 mg intravenously.

Maternal blood pressure, heart rate and axillary and midcalf skin temperatures were recorded every 5 minutes throughout the procedure. Oxygen saturation was monitored continuously by pulse oximetry using a finger probe; the oxygen saturation was not recorded unless it decreased to less than 95%. Shivering was assessed as follows: 0, no shivering; 1, mild observable shivering; 2, moderate and distressing shivering; 3, severe shivering, distressing to patient and interfering with monitoring of electrocardiography, pulse oximetry and/or blood pressure. The highest shivering score for each 5-minute interval was recorded and summated to give a cumulative shivering score for each

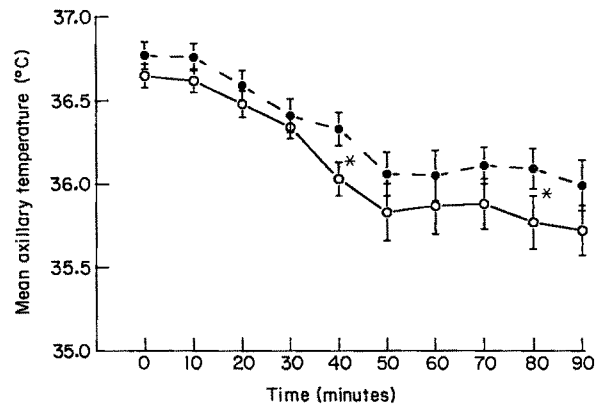


Fig. 2. The changes in the mean (SEM) axillary temperature in fentanyl (○—○) and placebo (●—●) groups, irrespective of shivering. (\*denotes  $p < 0.05$ , 0 minutes = when test dose was given).

patient. Blood loss was estimated from swab weights, suction bottle and sterile towels. 'Apgar minus colour' scores were assessed by the paediatrician at 1 and 5 minutes after delivery of the neonate.

Parametric data were analysed by one-way analysis of variance (ANOVA) and Student's *t*-test. Nonparametric data were analysed by Chi-squared-test with Yates' correction, Fisher's exact probability test or Wilcoxon rank test where appropriate. A probability value of  $< 0.05$  was considered to be statistically significant.

## Results

Forty-one parturients were studied. Eighteen were in the fentanyl group (group F) and 23 in the placebo group (group P). One parturient (in group F) shivered before the epidural catheter was inserted and another (in group P) had blood loss of more than one litre; data from these two subjects were excluded from subsequent analysis. There were no significant differences between the two groups with regard to age, weight, height, local anaesthetic dose, estimated blood loss, volume of fluid administered, ambient room temperature or duration of surgery (Table 1).

Shivering occurred in 21 out of 39 patients (55%), six in group F (35%) and 15 in group P (71%;  $p < 0.05$ ). There were two parturients with grade 3 shivering in group F and four in group P. The mean (SD) time of onset of shivering in group F was 11.7 (5.2) minutes, while that in group P

Table 1. Potential confounding variables in patients who received fentanyl (group F) or placebo (group P). Data presented as mean (SD).

	Group F	Group P
Age; years	28.5 (5.1)	26.8 (4.6)
Height; m	1.55 (0.09)	1.57 (0.06)
Weight; kg	71.5 (9.9)	68.9 (8.3)
Bupivacaine doses; mg	182.2 (6.5)	179.4 (7.2)
Intravenous fluid; litres	2.65 (0.38)	2.82 (0.42)
Blood loss; litres	0.58 (0.12)	0.66 (0.21)
Room temperature; °C	22.4 (0.3)	22.7 (0.2)
Duration of surgery; minutes	98.7 (8.0)	101.5 (13.2)

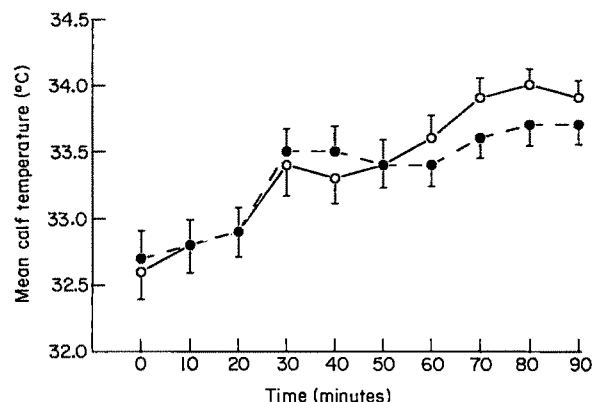


Fig. 3. The changes in the mean (SEM) calf temperature in fentanyl (○—○) and placebo (●—●) groups, irrespective of shivering. (0 minutes = when test dose was given).

was 21.5 (15.3) minutes. Shivering occurred within 90 minutes in all patients who shivered. The mean (SD) duration of shivering in group F was 52.5 (18.2) minutes and in group P 42.7 (20.4) minutes. There were no significant differences between the groups in either the time of onset or duration of shivering. The cumulative shivering scores in both groups during the first 90 minutes of the procedure are illustrated in Figure 1.

Figures 2 and 3 show the changes in mean axillary temperature and mean calf temperature in the fentanyl and placebo groups, regardless of the presence or absence of shivering. In general, it appeared that the parturients in group F had a consistently lower mean axillary temperature than those in group P, although the differences were significant only on two occasions. The fentanyl group also appeared to have a higher mean calf temperature in the later part of the procedure, but these differences were not significant.

Figure 4 shows the changes in the mean axillary temperature for the shiverers and nonshiverers in both the fentanyl and placebo groups. There were highly significant changes (ANOVA) within these four individual subgroups. However, it appeared that the fentanyl nonshiverers,

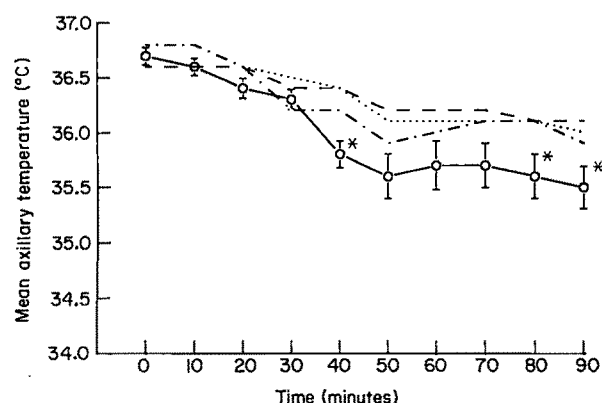


Fig. 4. The changes in the mean axillary temperature in fentanyl shiverers (---), fentanyl nonshiverers (○—○), placebo shiverers (.....) and placebo nonshiverers (-.-.-). The SEM of fentanyl nonshiverers is also indicated. (\*denotes  $p < 0.05$ , 0 minutes = when test dose was given).

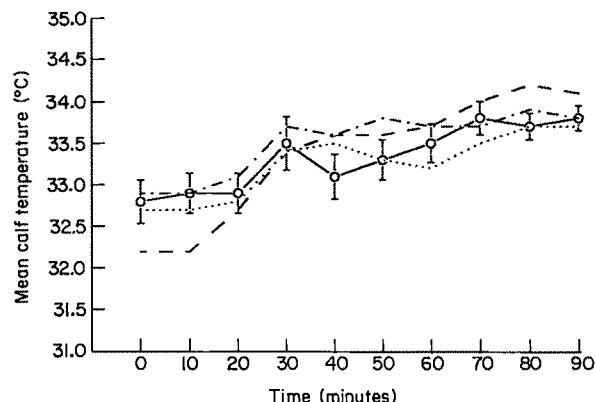


Fig. 5. The changes in the mean calf temperature in fentanyl shiverers (---), fentanyl nonshiverers (○—○), placebo shiverers (.....) and placebo nonshiverers (-.-.-). The SEM of fentanyl non-shiverers is also indicated. (0 minutes = when test dose was given).

compared to the other subgroups, had the largest decrease in mean axillary temperature; these changes were significant on three occasions ( $t$ -test). Figure 5 shows the changes in the mean calf temperature in each subgroup; the differences were not significant. Figure 6 shows the mean of the difference between axillary and calf temperatures in each subgroup over the 90-minute observation period; there were no differences between groups.

None of the parturients had oxygen saturations less than 95% at any time. Hypotension, defined as systolic arterial pressure less than 100 mmHg, occurred in six patients in group F and nine in group P (Table 2). The mean (SD) dose of ephedrine was 9.0 (4.2) mg in group F and 8.2 (3.5) mg in group P. Five subjects (three of whom complained of peritoneal pain) in group P and one in group F required further analgesic supplements. Four subjects in group F (one of whom also shivered) and two in group P complained of nausea and/or vomiting which required treatment with metoclopramide 10 mg. There was one case of pruritus in the fentanyl group. The 'Apgar minus colour' scores were 8 at 1 and 5 minutes for all neonates.

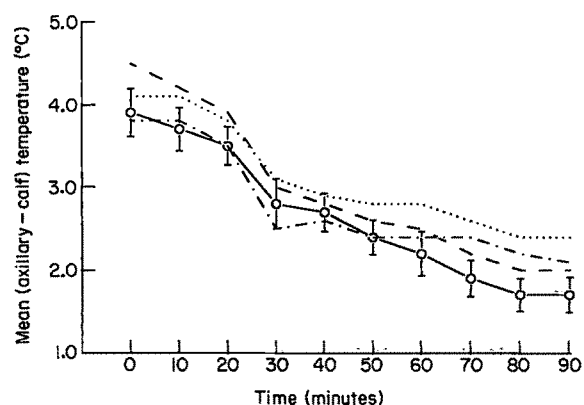


Fig. 6. The changes in the mean axillary and calf temperature differences in fentanyl shiverers (---), fentanyl nonshiverers (○—○), placebo shiverers (.....) and placebo nonshiverers (-.-.-). The SEM of fentanyl non-shiverers is also indicated. (0 minute = when test dose was given).

**Table 2.** Incidences of side effects and requirement for additional analgesic or antiemetic drugs, and mean (SD) dose of ephedrine required in hypotensive patients, after administration of fentanyl or placebo before epidural block.

	Fentanyl ( <i>n</i> = 17)	Placebo ( <i>n</i> = 22)
Hypotension	6	9
ephedrine, mg	9.0 (4.2)	8.2 (3.5)
Extra analgesia	1	5
Nausea	4	2
Antiemetic	4	2
Pruritus	1	0

### Discussion

Shivering in parturients during epidural analgesia is common. It has been reported to occur in up to 61% of parturients who receive epidural analgesia for Caesarean section.<sup>2</sup> There are several reasons why shivering during epidural analgesia should be avoided. Firstly, shivering not infrequently causes discomfort and distress to the mother. It has been reported that, of all the unwanted effects of epidural anaesthesia, shivering was the most disconcerting part of the birth experience.<sup>5</sup> Secondly, severe shivering may interfere with monitoring and may therefore compromise the safety of mother and fetus. Finally, while it remains to be proven that severe shivering may reduce oxygen availability to the fetus, it is known that severe shivering after general anaesthesia may increase total oxygen consumption fivefold, and may decrease arterial oxygen saturation.<sup>6</sup>

Attempts have been made to reduce the incidence of shivering by the use of warmed epidural injectate,<sup>7,8</sup> warmed intravenous fluid<sup>9</sup> or a combination of the two.<sup>10</sup> The results of these studies were conflicting. Recent evidence has suggested that shivering during epidural analgesia does not result solely from cooling of the epidural space in nonpregnant volunteers.<sup>11</sup> The administration of opioids such as pethidine,<sup>12</sup> fentanyl<sup>3</sup> or sufentanil<sup>13,14</sup> via the epidural route appears to provide more consistent results in the reduction of shivering during epidural analgesia. However, interpretation of the literature is complicated for the following reasons. Firstly, mothers in labour may respond differently in their thermoregulatory responses in comparison to nonpregnant controls.<sup>15</sup> Secondly, some studies included parturients in labour and those undergoing Caesarean section with epidural analgesia.

In this study, we were able to demonstrate a significant reduction of 50% in the incidence of shivering in patients treated with epidural fentanyl 25 µg given prophylactically. Factors which might have influenced changes of body temperature and/or thermoregulation, such as volume of intravenous fluid and doses of local anaesthetics administered, volume of blood loss and duration of surgery were not significantly different between the two groups. The overall incidence of shivering was 55%. This figure is comparable to the incidence of shivering reported in a previous study.<sup>2</sup> It has been suggested that the use of adrenaline itself may influence the incidence of shivering but the increased incidence was not significant in the original study.<sup>16</sup> Our data also showed that low-dose

epidural fentanyl did not delay the onset or shorten the duration of shivering. The incidences of unwanted effects in the two groups did not differ significantly. The incidence of hypotension and the requirement for ephedrine were similar. There was a slight increase in the incidence of nausea and/or vomiting in the fentanyl group. However, the fentanyl group appeared to have required less analgesic supplementation despite the low dose of fentanyl which was given. None of the neonates required resuscitation and there was no immediate or delayed respiratory depression in the mothers.

It has been suggested recently that hypothermia associated with epidural analgesia is due mostly to redistribution of body heat (i.e. decreases in core temperature and cutaneous temperature above the upper level of the block are accompanied by an increase in peripheral cutaneous temperature below this level) and not due to exaggerated heat loss to the surroundings.<sup>3</sup> However, it is not clear whether shivering is a result of hypothermia during epidural analgesia. Our data did not show any significant difference in the changes in the mean axillary or mid-calf temperatures between the placebo shiverers and non-shiverers. Other factors such as the preferential blockade of nerve fibres which conduct warm sensation,<sup>17</sup> or stimulation of thermoreceptors in the epidural space and spinal canal,<sup>7</sup> may have been more significant contributors.

It appeared from our observations that parturients who had fentanyl, regardless of the presence of shivering, tended to have a lower mean axillary temperature compared to those who had placebo, although the fentanyl nonshiverers might have contributed to most of these changes. However, the mean calf temperature in the fentanyl and the placebo groups was not significantly different. If shivering during epidural analgesia had been secondary to hypothermia, it may be speculated that fentanyl might have influenced the normal physiological response to hypothermia and therefore reduced shivering in these subjects. This is supported by the observation that opioids may interfere with thermoregulation by modulating nociceptive and temperature information at the spinal and/or higher levels in rodents.<sup>18</sup>

Epidural opioids given to parturients must be used with great care, and their use is perhaps limited by the potential risks of cardiorespiratory depression in the newborn and/or respiratory depression in the mother.<sup>19</sup> Nevertheless it has been demonstrated that fentanyl concentration in cord blood taken immediately after delivery is negligible (i.e. less than 0.1 ng/ml) after 100 µg fentanyl administered via the epidural route one hour prior to delivery.<sup>20</sup> Furthermore, it has been reported recently that fentanyl 100 µg administered epidurally before delivery significantly improves intra-operative comfort and the duration of postoperative analgesia.<sup>21</sup> However, it is not clear whether fentanyl administered epidurally at this higher dosage would reduce the incidence of shivering to an even greater extent than we achieved with a dose of 25 µg.

### Acknowledgments

The authors acknowledge the advice of Professor A. R. Aitkenhead on the preparation of the manuscript. The authors also acknowledge Mrs M. Lalloo and the staff on the Delivery Suite, the Pharmacy Department, and the staff in Obstetric Theatre in the University Hospital,

Nottingham for their unfailing support and cooperation during the study. This project was supported by a grant from BUPA Medical Foundation Limited.

### References

- BROWNRIDGE P. Shivering related to epidural blockade with bupivacaine in labour, and the influence of epidural pethidine. *Anaesthesia and Intensive Care* 1986; **14**: 412-7.
- MORGAN BM, AULAKH JM, BARKER JP, REGINALD PW, GOROSZENIUK T, TROJANOWSKI A. Anaesthetic morbidity following caesarean section under epidural or general anaesthesia. *Lancet* 1984; **1**: 328-30.
- MATTHEWS NC, CORSER G. Epidural fentanyl for shaking in obstetrics. *Anaesthesia* 1988; **43**: 783-5.
- SESSLER DI, PONTE J. Hypothermia during epidural anaesthesia results mostly from redistribution of heat within the body, not heat loss to the environment. *Anesthesiology* 1989; **71**: A882.
- OSTHEIMER GW, DATTA S. Observations in the postpartum recovery room after various local anaesthetic techniques. *Regional Anesthesia* 1981; **6**: 13-7.
- PRYS ROBERTS C. Post anesthesia shivering. In: ARTUSIO JF, ed. *Clinical anesthesia*. Philadelphia: F.A. Davis, 1968: 358.
- PONTE J, COLLETT BJ, WALMSLEY A. Anaesthetic temperature and shivering in epidural anaesthesia. *Acta Anaesthesiologica Scandinavica* 1986; **30**: 584-7.
- WEBB PJ, JAMES FM, WHEELER AS. Shivering during epidural anaesthesia in women in labor. *Anesthesiology* 1981; **55**: 706-7.
- WORKHOVEN MN. Intravenous fluid temperature, shivering, and the parturient. *Anesthesia and Analgesia* 1986; **65**: 496-8.
- MEHTA P, THERIOT E, MEHROTRA D. Shivering following epidural anesthesia in obstetrics. *Regional Anesthesia* 1984; **9**: 83-5.
- PONTE J, SESSLER DI. Extradurals and shivering: effects of cold and warm extradural saline injections in volunteers. *British Journal of Anaesthesia* 1990; **64**: 731-3.
- CASEY WF, SMITH CE, KATZ JM, O'LOUGHLIN K, WEEKS SK. Intravenous meperidine for control of shivering during Caesarean section under epidural anaesthesia. *Canadian Journal of Anaesthesia* 1988; **35**: 128-33.
- JOHNSON MD, SEVARINO FB, LEMA MJ, DATTA S, OSTHEIMER GW, NAULTY JS. Effect of epidural sufentanil on temperature regulation in the parturient. *Anesthesiology* 1987; **67**: A450.
- JOHNSON MD, SEVARINO FB, LEMA MJ. Cessation of shivering and hypothermia associated with epidural sufentanil. *Anesthesia and Analgesia* 1989; **68**: 70-1.
- ROLBIN SH. Editorial review of four papers on shivering. *Obstetric Anesthesia Digest* 1987; **7**: 64.
- SHEHABI Y, GATT S, BUCKMAN T, ISERT P. Effect of adrenaline, fentanyl and warming of injectate on shivering following extradural analgesia in labour. *Anaesthesia and Intensive Care* 1990; **18**: 31-7.
- BROMAGE PR. *Epidural analgesia*. Philadelphia: W.B. Saunders, 1978: 394-6.
- KATSUURA G, YOSHIKAWA K, ITOH S. Suppressive effect of beta-endorphin on body shaking response to ice water immersion and synergistic action of vasoactive intestinal peptide on beta-endorphin in anesthetized rats. *Japanese Journal of Physiology* 1983; **33**: 821-6.
- BROCKWAY MS, NOBLE DW, SHARWOOD-SMITH GH, MCCLURE JH. Profound respiratory depression after extradural fentanyl. *British Journal of Anaesthesia* 1990; **64**: 243-5.
- YOUNGSTROM P, EASTWOOD D, PATEL H, BHATIA R, COWAN R, SUTHEIMER C. Epidural fentanyl and bupivacaine in labor; double-blind study. *Anesthesiology* 1984; **61**: A414.
- KING MJ, BOWDEN MI, COOPER GM. Epidural fentanyl and 0.5% bupivacaine for elective Caesarean section. *Anaesthesia* 1990; **45**: 285-8.



## Monitoring of irrigating fluid absorption during transurethral prostatectomy

A study in anaesthetised patients using a 1% ethanol tag solution

J. HULTÉN, V. J. SARMA, H. HJERTBERG AND B. PALMQUIST

### Summary

*A simple, reliable method to detect absorption of irrigating fluid during transurethral prostatectomy is to tag irrigating fluids with 1% ethanol and monitor expired breath ethanol concentrations. This method correlated well ( $n = 0.79$ ) with other existing methods of absorption monitoring in 20 anaesthetised patients. Ethanol (1%) tagging does not alter the optical quality of the irrigating fluid and is harmless to the patient. The technique is non-invasive, repeatable, cheap and gives instant results. It can be used in anaesthetised or awake patients and can detect absorption of as little as 100–150 ml in any 10-minute period.*

### Key words

*Operation;* transurethral resection of the prostate.

*Complications;* irrigation fluid absorption.

*Monitoring;* ethanol.

Most patients who present for transurethral resection of the prostate (TURP) are elderly and have a high incidence of cardiopulmonary problems, high blood pressure, obesity and diabetes mellitus.<sup>1</sup> Severe cardiovascular, fluid and electrolyte disturbances and increased postoperative morbidity and mortality could result from a sudden absorption of about 1–3 litres of fluid. The mortality which arises from this operation has, however, decreased since the introduction of nonhaemolytic irrigating fluids<sup>2,3</sup> but symptoms from fluid absorption still occur in 5–10% of the patients.<sup>4–6</sup>

Under regional anaesthesia the patient complains characteristically of dizziness, headache, nausea, tightness in the chest and throat and shortness of breath. The symptoms may appear at any time during surgery or in the recovery room.<sup>7,8</sup> Under general anaesthesia the diagnosis of TURP syndrome is difficult and often delayed because the presenting signs are unexplained swings in blood pressure and severe refractory bradycardia; recovery from general anaesthesia and muscle relaxants may be delayed.<sup>9</sup> It is important that anaesthetists recognise absorption so that steps to limit it can be instituted. Recently we introduced a simple, noninvasive method of monitoring for irrigant fluid absorption.<sup>10</sup> Irrigant fluids are tagged with ethanol and expired breath ethanol (EB-ethanol) concentration is estimated using a breath alcohol analyser. The method was

tested on cooperating patients who underwent TURP under spinal or epidural anaesthesia.<sup>6,10–12</sup> Since some of our patients receive general anaesthesia, we decided to compare the EB-ethanol method with serial serum sodium estimations, and volumetric analysis of irrigant balance.

### Materials and methods

The Ethics Committees of the Universities of Umeå and Linköping, Sweden, approved the study, and 20 ASA grade 1 and 2 patients undergoing TURP for benign prostatic hypertrophy consented to participate; 10 were operated on at Piteå General Hospital and 10 at Norrköping General Hospital. A Storz 27 Fr resectoscope and the intermittent irrigating technique with glycine-ethanol (System Uromatic, Baxter, Sweden) containing 1.5% glycine and 1% ethanol (w/v) was used in all cases. On arrival in the operating theatre intravenous Ringer's acetate solution (sodium content 130 mmol/litre) was started.

All patients were anaesthetised and their lungs ventilated manually following a standard premedication. No volatile anaesthetic was used, and the end-tidal carbon dioxide, measured with an Engström Eliza Duo, was kept between 4.5 and 6.0 kPa. A radial artery catheter was inserted to allow blood sampling and pressure monitoring.

J. Hultén, MD, PhD, Consultant Urologist, V.J. Sarma, FFARCSI, Consultant Anaesthetist, Piteå General Hospital, 941 28 Piteå, Sweden, H. Hjertberg, MD, Consultant Urologist, B. Palmquist, MD, Consultant Anaesthetist, Norrköping General Hospital, Norrköping, Sweden.

Accepted 26 August 1990.

**Table 1.** Demographic details ( $n = 20$ ). Values expressed as mean (SD).

Age; years	68.6	(9.4)
Operation time; minutes	42	(15.08)
Resection weight; g	31.35	(21.25)
Blood loss; ml	555.8	(483.2)
Irrigant used; litres	10.14	(3.59)
Absorption; litres	0.552	(0.46)
Maximum EB-ethanol; %	0.155	(0.15)
Maximum decrease in serum Na; mmol/litre	4.4	(3.86)

### EB-ethanol estimation

Expired breath samples for ethanol concentration were drawn every 5 minutes with an Alcolmeter S-D2 (Lions Laboratories Ltd., Barry, Wales) through a modified end-tidal carbon dioxide connector attached to the tracheal tube (Piteå Hospital) and directly through a hole bored into the tracheal tube (Norrköping Hospital). This device measured the ethanol content of the exhaled breath and extrapolated the value to the corresponding blood ethanol concentration based upon the ratio of blood/EB-ethanol concentration<sup>13</sup> (one part per thousand (1‰) = 0.1 g/dlitre = 21.7 mmol/litre) in steps of 0.05‰ from 0 to 9.95‰. The Alcolmeter was calibrated after each use. Sampling was performed from the end-tidal breath which we found easier to obtain during manual ventilation. Measurements, if needed, were made well into the recovery period, every 5 minutes, until no alcohol was detectable in the expired breath.

### Volumetric fluid balance

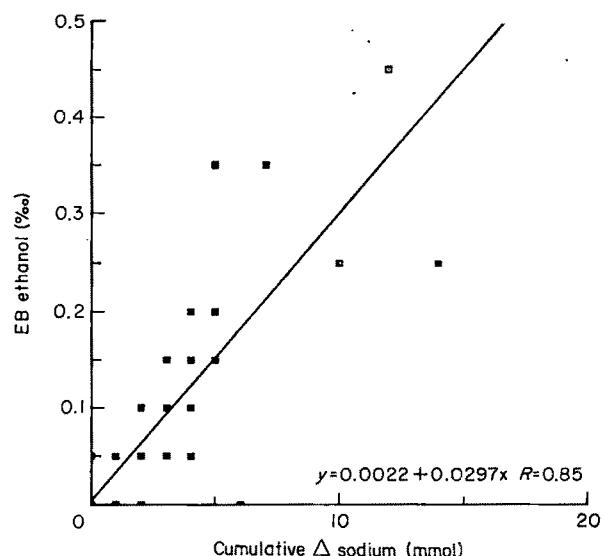
The volumetric fluid balance was calculated according to the method described by Hahn *et al.*<sup>14</sup> The irrigating fluid bags were weighed before and after 10 minutes' use. The difference in grammes was considered to be the volume in ml used. Every 10 minutes during the operative procedure the irrigant inlet was closed, the bladder emptied and the used irrigant bag and collecting bucket were replaced. The returned irrigant was measured volumetrically and the amount of blood in the returns was calculated with a Hemoglobin Photometer apparatus (Electrolux Megatronics, Leo AB, Helsingborg, Sweden). The amount of irrigant used was compared with the volume recovered, minus the calculated blood content. The difference between these volumes is the amount absorbed. No correction for urine excretion was made.

### Blood samples

Samples for the estimation of blood haemoglobin (B-Hb) and serum sodium (serum Na) were drawn every 10 minutes from the radial artery catheter as long as ethanol was detectable in the expired breath.

### Statistical analysis

Simple linear and stepwise multiple regression analysis was used and correlations were included when they were significant ( $p < 0.05$ ). Probability was measured by Student's *t*-test.



**Fig. 1.** Ethanol concentration in expired breath compared with the cumulative decrease in serum sodium concentration from the beginning of the operation. Serum Na was measured at the end of each 10-minute period during TURP together with estimations of EB-ethanol.

### Results

Patient data as regards age, resection time, resected weight and other details are presented in Table 1.

The 20 operations covered 80 10-minute periods. EB-ethanol (0.05%–0.45%) was detected in 32 periods and in all there was irrigant absorption, as determined by volumetric analysis. The lowest EB-ethanol detectable with an Alcolmeter (0.05%) was obtained in 10 periods. The mean volumetric absorption was 143 ml/10 minutes.

There was no ethanol in the expired breath during five operations (19 periods). In these patients, the mean absorption in 10 minutes calculated by volumetric method, was 38 ml and the mean absorption/operation was 146 ml.

A significant correlation was obtained between the EB-ethanol value at the end of a given collection period and the cumulative decrease in serum Na ( $r = 0.852$ ;  $p < 0.0001$ ) (Fig. 1). A comparable correlation was observed between EB-ethanol and the cumulative volume of irrigant absorbed from the start of the operation ( $r = 0.788$ ;  $p < 0.0001$ ) (Fig. 2). There was no correlation between the amount of irrigant absorbed and total blood loss, resected weight of prostate, resection time or the total amount of irrigant used. The selected correlations are presented in Table 2.

In some cases the absorption was mainly intravenous (IVA), characterised by a sharp increase and sudden decrease in EB-ethanol levels as the intravesical pressure is

**Table 2.** Significant correlations.

Cumulative decrease in serum Na compared with EB-ethanol	$r = 0.852$ ; $p < 0.0001$
Cumulative irrigant absorption compared with EB-ethanol	$r = 0.788$ ; $p < 0.0001$
Absorption in 10 minutes compared with EB-ethanol	$r = 0.705$ ; $p < 0.0001$
Cumulative drop in serum Na compared with cumulative absorption	$r = 0.695$ ; $p < 0.0001$

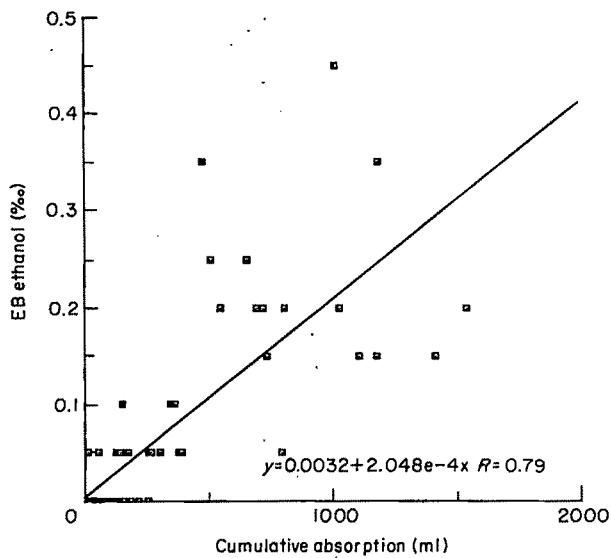


Fig. 2. Ethanol concentration in expired breath compared with the cumulative absorption of irrigant from the start of the operation.

reduced, accompanied by comparable changes in serum Na (Fig. 3(a)). In others the route of absorption was predominantly extravascular (EVA) when there was a gradual increase and decrease in EB-ethanol levels, even after emptying the bladder, together with a sustained drop in serum Na levels (Fig. 3(b)). In some cases there was a mixed type of absorption.

Before undertaking the study we tested the Alcolmeter with various concentrations of nitrous oxide, halothane, enflurane and isoflurane without obtaining a positive result.

### Discussion

Absorption of irrigant fluid through open veins in the prostatic bed is the primary cause of TURP syndrome during resection. A significant volume of irrigant can accumulate in the periprostatic and retroperitoneal spaces as well as be absorbed directly into the circulation. One series reported the incidence of extravascular absorption as 22%.<sup>15</sup> Oester and Madsen<sup>16</sup> have claimed that about two-thirds of the irrigant volume is absorbed extravascularly.

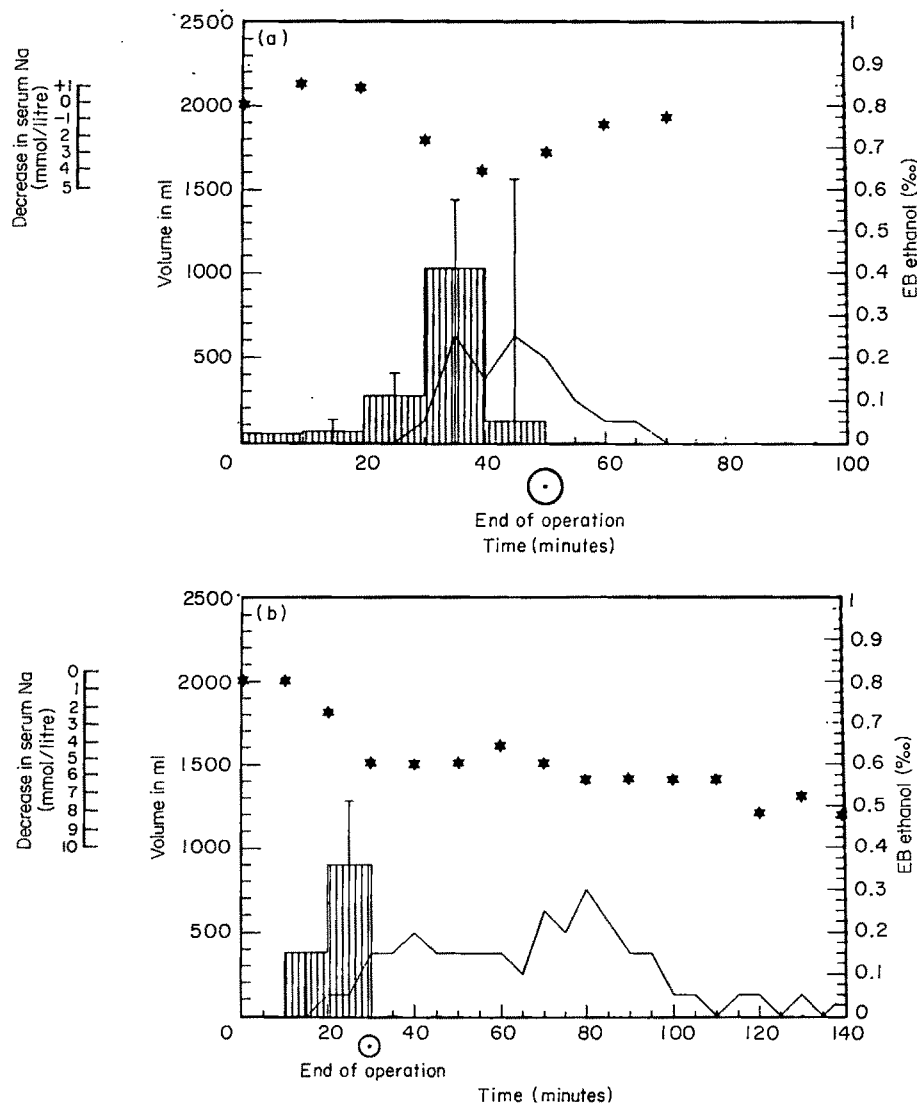


Fig. 3 (a) and (b). Schematic representation of irrigant absorption as assessed by three different methods during TURP. (a) Reflects predominantly intravenous absorption and (b) shows extravascular absorption events. ★, Serum-Na; ▮, cumulative absorption; ▨, absorption in 10 minutes; —, EB-ethanol.

This is all too common when the prostatic capsule is breached during surgery. The other important factor that determines the rate of irrigant absorption is the hydrostatic pressure in the prostatic bed.<sup>17</sup> The duration of resection, contrary to popular belief, has no relationship to the development of TURP syndrome. Melchior *et al.*, after an analysis of 2223 consecutive TURP operations, concluded that absorption is time related, only in resections which exceeded 150 min,<sup>18</sup> while excessive absorption leading to coma has been reported within 15–20 minutes of the start of surgery.<sup>7,19</sup> It is therefore of interest to the anaesthetist to have a simple, reliable, noninvasive method of irrigant fluid absorption monitoring.

We discovered that the intact bladder mucosa was impermeable to 2% alcohol contained in the irrigating fluids.<sup>10</sup> We then tagged irrigating fluids with 2% ethanol and measured the EB-ethanol, assuming that the presence of ethanol in the expired breath could only have occurred as a result of absorption. Subsequent studies<sup>6,11,12</sup> have shown that this technique is reliable in patients who receive spinal and epidural anaesthesia. Under general anaesthesia the early warning signs of TURP syndrome are masked, and therefore serious absorption can occur before haemodynamic and ECG changes are seen.

The Alcolmeter used in this study is accurate, specific for alcohol<sup>13</sup> and, unlike infrared analysers, is not influenced by the presence of anaesthetic gases.<sup>20</sup> Alcohol levels can be monitored every 2 minutes if desired. Using 1% ethanol-tagged irrigating fluids, absorption of 100–150 ml/10-minute period can be detected. In a previous study<sup>11</sup> we suggested a simple formula for the estimation of the absorbed volume. Absorption (litre) = 3.6 EBV-ethanol (max)/Ethanol concentration in % in the irrigating fluid ( $r = 0.89$ ;  $p < 0.001$ ).

Absorption of as little as 50 ml/10 minutes could be detected when irrigant fluids containing 2% ethanol were used.<sup>11</sup> We reserve 2% ethanol solution for cases where it is especially important to detect the absorption of small amounts e.g. high-risk patients and when plain water is used as irrigating solution (in order to avoid haemolytic complications). Volumetric irrigating fluid balance and serum Na estimations are accurate but cumbersome; the results are not obtained instantly and trends are difficult to follow. They are, moreover, invasive and labour intensive. The volumetric method cannot be followed into the post-operative period and gives no information about the route of absorption, intravascular or extravascular.

Radioactive isotopes have been used to tag irrigating fluids<sup>16</sup> and when accurately monitored, this method gives a continuous, relatively accurate record of absorption events. It requires, however, costly and bulky equipment, trained personnel and there is the problem of radioactive waste disposal after each operation. Hjertberg *et al.*<sup>21</sup> compared the EB-ethanol method with continuous radioisotope estimations by simultaneously tagging irrigating fluids with Technetium 99 m and 2% alcohol. They found a significant linear relationship between the two methods ( $r = 0.91$ ) and also between each method when compared with volumetric estimation of irrigant absorption. Ethanol is cheap and harmless in the quantities used and was well tolerated by the patients. It does not alter the optical characteristics of the irrigant fluid and is nonelectrolytic, so poses no problems for the resectionist.

The spread of values around the regression line in

Figure 2 reflects the complicated dynamics involved during absorption. Absorption is influenced by many variables such as intravesical pressure, damaged veins, breached prostatic capsule and the timing of sampling. We obtained, however, better correlation between EB-ethanol and the cumulative volume of absorbed irrigant ( $r = 0.788$ ) compared with the decrease in serum Na and the cumulative absorption ( $r = 0.695$ ). This leads us to believe that EB-ethanol monitoring is more accurate than the serum Na method of irrigant absorption. Ethanol is rapidly distributed in the total body water, so a rising EB-ethanol value implies that irrigant is being absorbed and a steady level indicates that irrigant is being absorbed at the same rate as ethanol is leaving the circulation. A decrease in EB-ethanol values is seen when the rate of absorption has decreased or ceased.

It is also possible to identify the route of absorption by emptying the bladder and estimating the EB-ethanol for 2 minutes. A sharp decrease in EB-ethanol denotes an intravascular absorption, and a minimal or gradual decline indicates an extravascular absorption or a mixed type.

Information that absorption is occurring is valuable if steps can be taken to correct the situation. We inform the surgeon when the EB-ethanol level reads 0.3%, which is approximately equivalent to an absorption of about 1000 ml/10 minutes. The surgeon either reduces the intravesical pressure to limit absorption or cuts short the resection before the patient is seriously harmed.

It is also possible to differentiate between intra-operative absorption and anaesthesia-related events. This is important from the medicolegal aspect when the conduct of the anaesthetic is called into question. Pulmonary disease can lower the blood/breath coefficient of ethanol during expiration, although it has been reported that it does not affect end-tidal breath analysis.<sup>22</sup> The ethanol concentration can, however, vary slightly (SD 2.6%) as a result of inter and intra subject variations in breath temperature.<sup>23</sup>

Serious absorption has been reported following vesical ultrasonic lithotripsy,<sup>24</sup> intra-uterine endoscopic laser surgery<sup>25</sup> and percutaneous pyelolithotripsy.<sup>26</sup> We believe that this monitoring technique has applications in detecting fluid absorption in any operation where large volumes of fluids are introduced into body cavities. EB-ethanol monitoring during TURP provides the anaesthetist with a simple, safe, relatively exact method of detecting irrigant absorption. The morbidity and mortality associated with this operation can be reduced by heeding the early warning signal provided by this method.

### Acknowledgments

The authors wish to thank Dr G. Sundström in particular, for his help with the statistical aspects of this study and the nursing staff in the operating theatres and postoperative wards at Piteå and Norrköping General Hospitals for their cooperation.

### References

1. AZAR I. Transurethral prostatectomy syndrome. In: BARASH PD, (ed). The American Society of Anaesthesiologists Inc. *Refresher Courses in Anesthesiology* 1989; Vol. 17: 1–15.
2. CREEVY CD. Hemolysis and transurethral resection. *Surgery* 1956; **39**: 180–8.

3. EMMETT JL, GILBAUGH JH, MCLEAN P. Fluid absorption during transurethral resection: comparison of mortality and morbidity after irrigation with water and non-hemolytic solutions. *Journal of Urology* 1969; **101**: 884-9.
4. HARRISON RH III, BOREN JS, ROBISON JR. Dilutional hyponatremic shock: another concept of the transurethral prostatic resection reaction. *Journal of Urology* 1956; **75**: 95-110.
5. RHYMER JC, BELL TJ, PERRY KC, WARD JP. Hyponatraemia following transurethral resection of the prostate. *British Journal of Urology* 1985; **57**: 450-2.
6. HJERTBERG H, JORFELDT L, SCHELIN S. The use of ethanol as a marker substance to increase patient safety during transurethral prostatic resection. A screening investigation of absorption of irrigating fluid in four hospitals and a comparison between experienced and inexperienced Urologists. *Urology* (In press).
7. Hurlbert BJ, Wingard DW. Water intoxication after 15 minutes of transurethral resection of the prostate. *Anesthesiology* 1979; **50**: 355-6.
8. STILL JA, MODELL JH. Acute water intoxication during transurethral resection of the prostate, using glycine solution for irrigation. *Anesthesiology* 1973; **38**: 98-9.
9. ROESCH RP, STOELTING RK, LINGEMAN JE, KAHNOSKI RJ, BACKES DJ, GEPHARDT SA. Ammonia toxicity resulting from glycine absorption during transurethral resection of the prostate. *Anesthesiology* 1983; **58**: 577-9.
10. HULTÉN JO, JORFELDT LS, WICTORSSON YM. Monitoring fluid absorption during TURP by marking the irrigating solution with ethanol. *Scandinavian Journal of Urology and Nephrology* 1986; **20**: 245-51.
11. HULTÉN JO, HAHN RG. Monitoring irrigating fluid absorption during transurethral resection of the prostate (TURP); a comparison between 1 and 2% ethanol as a tracer. *Scandinavian Journal of Urology and Nephrology* 1989; **23**: 103-8.
12. HAHN RG. Ethanol monitoring of irrigating fluid absorption in transurethral prostatic surgery. *Anesthesiology* 1988; **68**: 867-73.
13. GIBB KA, YEE AS, JOHNSTON CC, MARTIN SD, NOWAK RM. Accuracy and usefulness of a breath alcohol analyzer. *Annals of Emergency Medicine* 1984; **13**: 516-20.
14. HAHN R, BERLIN T, LEWENHAUPT A. Irrigating fluid absorption and blood loss during transurethral resection of the prostate studied with a regular interval monitoring (RIM) method. *Scandinavian Journal of Urology and Nephrology* 1988; **22**: 23-30.
15. MASLOFF JL, MILAM JH, BUNTS RC. Fluid and electrolyte changes associated with transurethral prostatic resection. *Southern Medical Journal* 1966; **59**: 1203-8.
16. OESTER A, MADSEN PO. Determination of absorption of irrigating fluid during transurethral resection of the prostate by means of radioisotope. *Journal of Urology* 1969; **102**: 714-19.
17. HULTÉN JO. Prevention of irrigating fluid absorption during transurethral resection of the prostate. Doctoral thesis. Linköping University Medical Dissertations No. 176. Linköping University, Linköping, Sweden 1984: 15-43.
18. MELCHIOR J, VALK WL, FORET JD, MEBUST WK. Transurethral prostatectomy: computerised analysis of 2,223 consecutive cases. *Journal of Urology* 1974; **112**: 634-42.
19. HENDERSON DJ, MIDDLETON RG. Coma from hyponatremia following transurethral resection of the prostate. *Urology* 1980; **15**: 267-71.
20. GUYTON D, GRAVENSTEIN N. Alcohol interference with infrared anesthetic gas analysis. *Anesthesiology* 1989; **71**: A465.
21. HJERTBERG H, EKBERG S, HAHN R, HULTÉN J, JORFELDT L, SVEDBERG J. Irrigating fluid absorption during transurethral prostatic resection as measured by ethanol, radioisotopes and regular-interval monitoring. *Urology* (In press).
22. HAAS H, MORRIS JF. Breath-alcohol analysis in chronic bronchopulmonary disease. *Archives of Environmental Health* 1972; **25**: 114-18.
23. JONES AW. Quantitative measurements of the alcohol concentration and the temperature of breath during a prolonged exhalation. *Acta Physiologica Scandinavica* 1982; **114**: 407-12.
24. BATRA YK, KAPOOR R, HEMAL AK, VAIDYANATHAN S. Hyponatraemia and mental symptoms following vesical ultrasonic lithotripsy. *Anaesthesia* 1988; **43**: 675-6.
25. VAN BOVEN MJ, SINGELYN F, DONNEZ J, GRIBOMONT BF. Dilutional hyponatremia associated with intrauterine endoscopic laser surgery. *Anesthesiology* 1989; **71**: 449-50.
26. SINCLAIR JF, HUTCHISON A, BARAZA R, TELFER ABM. Absorption of 1.5% glycine after percutaneous ultrasonic lithotripsy for renal stone disease. *British Medical Journal* 1985; **291**: 691.

## Plasma concentrations of bupivacaine after supraclavicular brachial plexus blockade in patients with chronic renal failure

A. S. C. RICE, C. E. PITHER AND G. T. TUCKER

### Summary

*The plasma concentrations of bupivacaine and the latency and duration of anaesthesia after supraclavicular block with 30 ml of 0.5% bupivacaine were measured in 10 patients with chronic renal failure and in 10 patients with normal renal function. No significant difference was found between the two groups in respect of pharmacokinetic parameters, or in block latency or duration.*

### Key words

*Anaesthetic techniques, regional; brachial plexus block.*

*Anaesthetics, local; bupivacaine.*

*Pharmacokinetics.*

*Complications; renal failure.*

Brachial plexus block is an ideal technique for the provision of anaesthesia of the arm for the formation of fistulae in chronic renal failure (CRF).<sup>1</sup> The resulting sympathetic blockade provides optimal surgical conditions and general anaesthesia is avoided. Systemic toxicity would not be anticipated as a special risk in patients with compromised renal function since modern local anaesthetics are metabolised in the liver and are not reliant upon renal function for their elimination from the body.

There is, however, a clinical suspicion that brachial plexus block is less effective in patients with CRF than in those with normal renal function. This is supported by a study which showed a decreased duration (38%) of brachial plexus anaesthesia in CRF.<sup>2</sup> These workers<sup>2</sup> however, were unable to correlate this change with haematological or biochemical factors. It was suggested that it might reflect a faster systemic uptake of drug because of an increased cardiac output in renal failure.

In addition, reports of toxicity in CRF<sup>3,4</sup> have led to suggestions that the pharmacokinetics of local anaesthetics may be altered unfavourably in this condition, although conclusive data in this area are lacking. Gould and Aldrete<sup>4</sup> reported a patient in CRF who had five brachial plexus blocks. Three of these were uneventful but a fourth was complicated by CNS toxicity. All of these blocks were performed when the patient had normal plasma potassium concentrations and no metabolic acidosis. A fifth block was performed in the presence of hyperkalaemia and a meta-

bolic acidosis and cardiac toxicity occurred. However, their claim that bupivacaine toxicity is enhanced by the acidosis of CRF cannot be fully accepted since they were using very high doses of bupivacaine without adrenaline (250–300 mg) on the two occasions when toxicity occurred (as opposed to the three other events) which may well have caused toxic symptoms in the absence of acidosis or hyperkalaemia.

We have compared the characteristics of neural blockade and the plasma concentrations of bupivacaine after supraclavicular brachial plexus injections in patients with CRF and those with normal renal function.

### Methods

Local ethics committee approval was obtained before the start of the study. Ten patients with CRF, arising from a variety of causes, undergoing A-V fistula construction and 10 normal controls undergoing hand surgery gave written consent to take part in the study. Supraclavicular brachial plexus block was performed using a paraesthesia technique. Bupivacaine hydrochloride (30 ml 0.5%) was injected over 2 minutes. Venous blood was sampled through an indwelling cannula in the contralateral arm at 0, 7.5, 10, 15, 30, 45 and 60 minutes and at 2, 4 and 8 hours after injection. The plasma was separated and stored at  $-20^{\circ}\text{C}$  until analysis. Bupivacaine was assayed by gas chromatography with a nitrogen detector according to the method described by Mather and Tucker.<sup>5</sup> Serum concentrations of

A.S.C. Rice, FFARCS, Research Fellow, C.E. Pither, FFARCS, Consultant, G.T. Tucker,\* PhD, Professor, Department of Anaesthetics, St Thomas' Hospital, Lambeth Palace Road, London SE1 7EH and \*Department of Medicine and Pharmacology, University of Sheffield, Royal Hallamshire Hospital, Sheffield S10.

Correspondence should be addressed to Dr Pither please.



**Table 1.** Results of serum biochemical analysis and block characteristics (mean block latency and duration) after injection of bupivacaine (30 ml 0.5%) by the supraclavicular route in patients with chronic renal failure (CRF) ( $n=10$ ) and in patients with normal renal function (control) ( $n=10$ ).

	Control (mean/range)	CRF (mean/range)
<i>Serum biochemistry</i>		
Creatinine; $\mu\text{mol/litre}$	77 (54–105)	788 (327–1222)**
Urea; $\text{mmol/litre}$	4.5 (3.6–5.1)	26.7 (13.9–41.2)**
Total protein; $\text{g/litre}$	72 (69–76)	63.8 (57–79)
Albumin; $\text{g/litre}$	44 (41–51)	38 (32–43)
Alpha-1-acid glycoprotein; $\text{g/litre}$	0.86 (0.49–1.37)	1.28 (0.96–1.98)*
<i>Block latency; minutes</i>		
Motor	18.1 (5–35)	13.8 (10–25)
Sensory	15 (5–30)	15.6 (10–30)
<i>Block duration; hours</i>		
Paraesthesiae	6.9 (0.4–23.15)	4.4 (3–5.5)
Pain	11.8 (3.05–26.15)	11 (7.05–14.2)
Normal	17.9 (4.55–27.15)	12 (3.3–16.10)

Unpaired  $t$ -test \*significant; \*\*Very significant.

alpha-1-acid glycoprotein were measured using an immuno-diffusion technique. The area under the plasma bupivacaine concentration-time curve (AUC) up to the last data point was calculated using the linear trapezoid rule. Values of maximum plasma drug concentration ( $C_{\text{max}}$ ) and time to its occurrence ( $T_{\text{max}}$ ) were taken directly from the data.

Motor function and sensation in the area supplied by the brachial plexus were measured every 5 minutes until the block was fully established. The sensory latency was calculated as the time (from injection) at which 50% of the dermatomes eventually anaesthetised were insensitive to a blunt needle. Motor latency was assessed similarly, with complete block defined as total loss of power in the muscle groups tested.

Block duration was assessed using a questionnaire eliciting: times taken to return of paraesthesiae, pain and normality, after injection.

Demographic data, block characteristics and biochemical data were compared using Student's unpaired  $t$ -test. Plasma drug concentrations were compared using the Wilcoxon test. Confidence intervals for differences between means for unpaired data were calculated according to Gardner and Altman.<sup>6</sup>

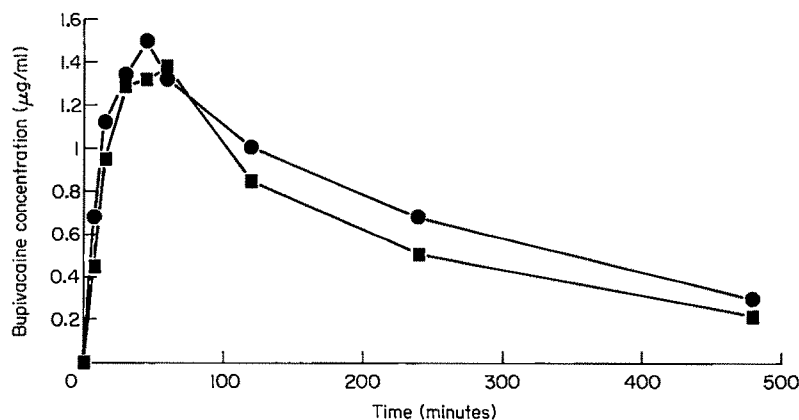
## Results

The two groups did not differ significantly in age or weight. The mean age (range) was 41.3 (20–67) years in the CRF group and 39 (24–59) years in the control group. The mean weights (range) were 70 (54–93) kg in the CRF group and 77.06 (58–111) kg in the control group. There was no difference in the sex distribution of the two groups.

All patients except one in the control group developed a block sufficient for surgery.

Serum creatinine, urea and alpha-1-acid glycoprotein concentrations were significantly higher in the CRF patients, but there were no significant differences between the groups with respect to serum albumin or total protein concentrations (Table 1).

There were no significant differences between the groups with respect to block characteristics (Table 1) or plasma drug concentrations (Figs 1, 2 and 3). The difference between the sample mean AUCs in chronic renal failure and normal patients was  $49.7 \mu\text{g/ml/minute}$ , with a 95% confidence interval from  $-71.5$  to  $170.9 \mu\text{g/ml/minute}$ . The difference between the sample mean  $C_{\text{max}}$  values was  $0.01 \mu\text{g/ml}$ , with a 95% confidence interval from  $-1.37$  to  $1.39 \mu\text{g/ml}$ .



**Fig. 1.** Mean plasma drug concentrations after injection of bupivacaine (30 ml 0.5%) by the supraclavicular route in patients with chronic renal failure (—●—, CRF) ( $n=10$ ) and in patients with normal renal function (—■—, control) ( $n=10$ ). Mean AUC ( $\mu\text{g/ml/minute}$ ): control 309 (169), renal failure 359 (103); mean  $C_{\text{max}}$  ( $\mu\text{g/ml}$ ): control 1.57 (0.78), renal failure 1.56 (0.51); median  $T_{\text{max}}$  (minutes): control 45 (30–60), renal failure 45 (15–60).

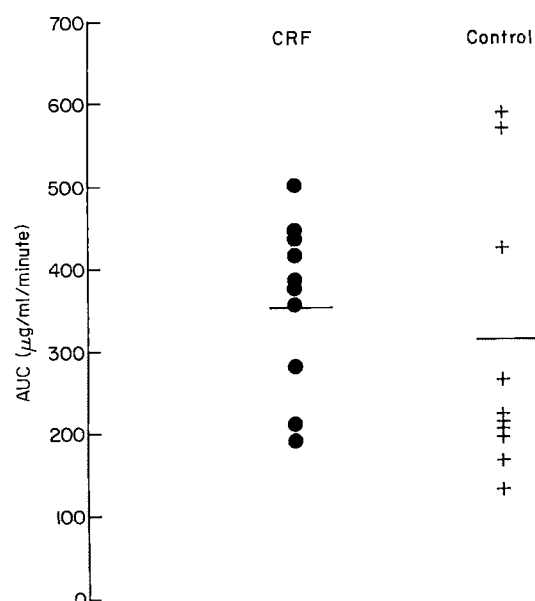


Fig. 2. Distributions of individual AUC (plasma bupivacaine concentration) values in patients with chronic renal failure (●, CRF) and in patients with normal renal function (+, control). (Bars indicate mean values).

### Discussion

In contrast to Bromage and Gertel<sup>2</sup> who reported that CRF was associated with a shorter duration of supraclavicular block, we did not detect any differences in block characteristics. It is possible that the statistical power of our study may have been insufficient to detect a small difference, but others have also not been able to replicate the original findings for bupivacaine when given by the axillary<sup>7</sup> or interscalene routes.<sup>8</sup> It should be emphasised that the study of Bromage and Gertel<sup>2</sup> used retrospective controls and an inadequate method of measuring block

duration. An adequate duration of block is important in patients undergoing A-V fistula formation since provision of analgesia and sympathetic blockade into the post-operative period can prevent arterial spasm and graft thrombosis. The accurate measurement of duration of brachial plexus blockade is difficult when a block can last up to 20 hours. We used a self-reporting method, which examined three different criteria separately rather than a single index or a choice of criteria as in other studies.<sup>2,7</sup>

The observation of similar plasma concentrations of bupivacaine in our patients with CRF and in those with normal renal function is not consistent with the suggestion that systemic uptake of local anaesthetic may be faster in CRF patients, who have a hyperdynamic circulation.<sup>2</sup> The increased serum concentration of alpha-1-acid glycoprotein, the major binding protein for bupivacaine, in the patients with CRF is compatible with the observations of others<sup>7,9</sup> and would suggest that, if anything, plasma concentrations of the free drug would be lower in this group, implying a lower risk of systemic toxicity. Although acidemia, associated with CRF, would have the opposite effect of decreasing the extent of bupivacaine binding<sup>10,11</sup> it seems unlikely that altered kinetics contribute significantly to the apparent enhanced toxicity of bupivacaine described in some case reports.<sup>3,4</sup>

Our findings on plasma drug concentrations confirm those of Martin *et al.*<sup>7</sup> and McEllistrem *et al.*<sup>8</sup> with lignocaine. These authors also report no differences in AUC and  $C_{max}$  for total drug in plasma between patients with CRF and those with normal renal function. Although Chauvin *et al.*<sup>12</sup> found a significantly lower mean  $C_{max}$  of bupivacaine in CRF patients compared to controls after axillary brachial plexus block, free drug concentrations were similar in the two groups.

We consider that brachial plexus blockade with bupivacaine is an ideal anaesthetic technique for A-V fistula surgery in patients with CRF. Our data indicate that these patients show similar block characteristics and pharmacokinetics to normal patients. However, the role of acidemia and hyperkalemia, seen in uncontrolled CRF, with respect to nerve block and systemic toxicity warrants further investigation.

### References

1. URBAN J, McDONALD H, STEEN S. Axillary block and dialysis. *Journal of the American Medical Association* 1967; **199**: 139-41.
2. BROMAGE PR, GERTEL M. Brachial plexus anesthesia in chronic renal failure. *Anesthesiology* 1972; **36**: 488-93.
3. EDDE R, DEUTSCH S. Cardiac arrest after interscalene brachial plexus block. *Anesthesia and Analgesia* 1977; **56**: 446-7.
4. GOULD D, ALDRETE J. Bupivacaine cardiotoxicity in a patient with renal failure. *Acta Anaesthesiologica Scandinavica* 1983; **27**: 18-21.
5. MATHER LE, TUCKER GT. Meperidine and other basic drugs: General method for their detection in plasma. *Journal of Pharmaceutical Sciences* 1974; **63**: 306-7.
6. GARDNER MJ, ALTMAN DG. *Statistics with confidence—confidence intervals and statistical guidelines*. London: British Medical Journal 1989. 21-2.
7. MARTIN R, BEAUREGARD L, TETRAULT J. Brachial plexus blockade and chronic renal failure. *Anesthesiology* 1988; **69**: 405-6.
8. MCELLISTREM RF, SCHELL J, O'MALLEY K, O'TOOLE D, CUNNINGHAM AJ. Interscalene brachial plexus blockade with lidocaine in chronic renal failure—a pharmacokinetic study. *Canadian Journal of Anaesthesia* 1989; **36**: 59-63.

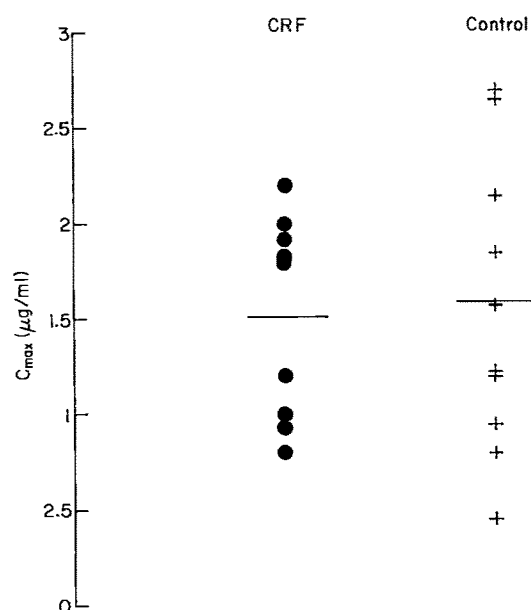


Fig. 3. Distributions of individual  $C_{max}$  (plasma bupivacaine) values in patients with chronic renal failure (●, CRF) and in patients with normal renal function (+, control). (Bars indicate mean values).

9. GROSSMAN S, DAVIS D, KITCHELL B, SHAND D, ROUTLEDGE P. Diazepam and lidocaine plasma protein binding in renal disease. *Clinical Pharmacology and Therapeutics* 1982; **31**: 350.
10. BURNEY R, DIFAZIO C, FOSTER J. Effects of pH on protein binding of lidocaine. *Anesthesia and Analgesia* 1978; **57**: 478-80.
11. SJÖSTRAND U, WIDMAN B. Distribution of bupivacaine in the rabbit under normal and acidotic conditions. *Acta Anaesthesiologica Scandinavica* 1973; **50** (Suppl.): 11.
12. CHAUVIN M, MAZOIT J, ANDRIVON F, GOEAU O, DUVALDESTIN P. Bupivacaine pharmacokinetics during axillary brachial plexus block in renal failure. *Anesthesiology* 1987; **67**: A261.

## The Brain laryngeal mask

A comparative study with the nasal mask in paediatric dental outpatient anaesthesia

R. BAILIE, M. B. BARNETT AND J. F. FRASER

### Summary

*Fifty ASA grade 1 children, who presented for dental outpatient extraction were studied. They were randomly allocated to two groups after induction: group 1 had conventional nasal mask anaesthesia and group 2 anaesthesia with a laryngeal mask. Group 2 had fewer hypoxic episodes and significantly better arterial oxygen saturations ( $p < 0.01$ ). There was no difference between the groups as regards surgical access, difficulty of extraction or bleeding. The laryngeal mask appears to provide an alternative to conventional nasal mask anaesthesia, with better overall oxygenation and would seem particularly suitable for prolonged or difficult extractions.*

### Key words

*Anaesthesia; dental, outpatient.  
Equipment; laryngeal mask.*

Outpatient paediatric dental anaesthesia requires the provision of an adequate level of anaesthesia, an unobstructed airway and protection from aspiration of blood and debris whilst allowing the operator adequate access within the oral cavity. Traditionally this has been achieved by the use of a nasal mask and packing.

In a number of studies<sup>1–3</sup> with this method almost 50% of patients demonstrated a significant degree of hypoxia. In a 10-year review of mortality associated with dental surgery,<sup>4</sup> 50% of deaths were associated with airway problems and it has been postulated that some degree of airway obstruction is inevitable.<sup>5</sup> The Brain laryngeal mask airway (LMA)<sup>6</sup> has been used to provide good airway control in adults<sup>7</sup> and has been successfully used for intra-oral procedures in children.<sup>8</sup>

The aim of this study was to determine whether the laryngeal mask could be used to provide a viable alternative for airway control for paediatric dental surgery.

### Method

Fifty patients, ASA grade 1, below the age of 16 years, requiring outpatient dental extraction under general anaesthesia were studied following local ethics committee approval. Those requiring single extractions or upper incisors only were excluded.

All patients were induced according to individual preference either with methohexitone 1.5 mg/kg or with

halothane in 30% oxygen with nitrous oxide. Maintenance was with halothane in 30% oxygen with nitrous oxide delivered from a Bain type coaxial breathing system to a facemask. Patients were randomly allocated to two groups according to hospital number: group 1 nasal mask anaesthesia and group 2 laryngeal mask anaesthesia.

When anaesthesia was considered sufficiently deep on clinical grounds in group 1, the facemask was changed for a nasal mask and anaesthesia continued with 30% oxygen in nitrous oxide with halothane, as clinically indicated. In group 2 the LMA was inserted when anaesthesia was judged sufficiently deep. Anaesthesia was continued with halothane as clinically indicated until the end of the procedure. The airway was left *in situ* until reflexes had returned.

All patients had continuous ECG and  $SaO_2$  monitoring from immediately after induction to the end of the procedure. End-tidal  $CO_2$  was monitored using a Cardiacap in those patients with a laryngeal mask. Pulse rate, oxygen saturation and, where applicable, end-tidal  $CO_2$  were recorded at 30-second intervals. The time of induction of anaesthesia, introduction of airway or nasal mask and start and end of surgery were all recorded. Specific intra-operative events such as episodes of obstruction and occurrence of arrhythmias were recorded, together with surgical activity at the time.

In all cases the surgeon was asked to grade the degree of difficulty in extraction and ease of access on a scale of 1

R. Bailie, MB, FFARCS(I), Senior Registrar, M.B. Barnett, FFARCS, Consultant, Department of Anaesthetics, Guy's Hospital, London SE1 9RT, J.F. Fraser, BDS, House Officer, Department of Oral Surgery, Guy's Hospital, London SE1 9RT.

Accepted 21 August 1990.

**Table 1.** Demographic data.

	Group 1	Group 2
Mean age; years (SD)	6.08 (2.34)	5.60 (2.32)
Weight; kg (SD)	23.2 (6.51)	21.15 (4.95)
Median number of extractions (range)	5 (2–16)	6 (2–13)

**Table 2.** Mean total anaesthetic time and surgical time in minutes (SD).

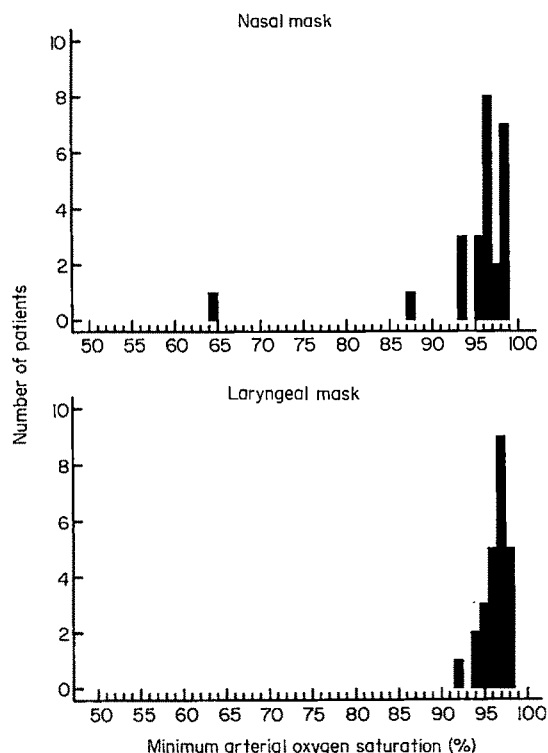
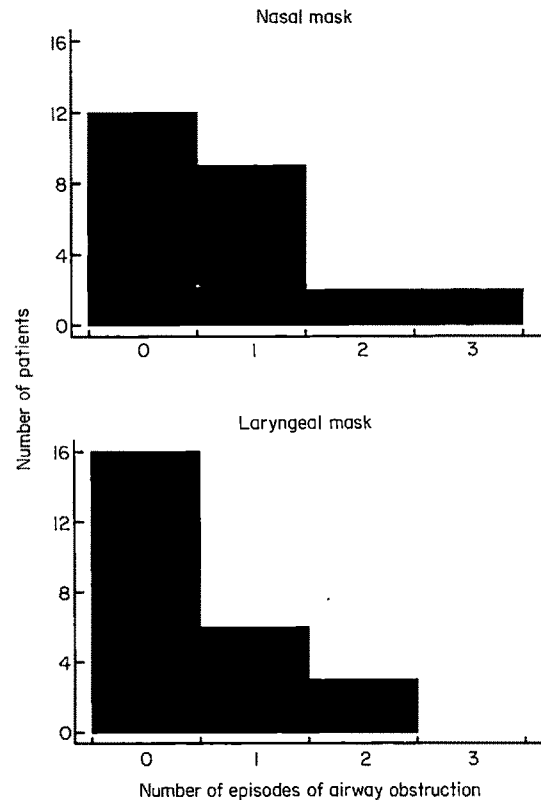
	Group 1	Group 2	p value
Anaesthetic time	7.75 (2.22)	9.31 (3.12)	0.07
Surgical time	2.84 (1.62)	4.02 (1.98)	0.02
Surgical/anaesthetic %	36.64	43.18	

(easy, excellent) to 5 (very difficult, very poor). At the end of surgery the amount of blood on the distal end of the pack was graded on a scale from 1 (very little) to 3 (soaked).

Results were analysed using Student's *t*-test, Chi-squared and Mann–Whitney *U*-tests as appropriate.

### Results

Demographic data for the two groups are summarised in Table 1. There was no significant difference between the two groups. Table 2 shows total anaesthetic and surgical time for the two groups and surgical time as a percentage of anaesthetic time. Surgical time was significantly longer ( $p = 0.02$ ) in the LMA group and was also a larger proportion of anaesthetic time.

**Fig. 1.** Histogram showing the frequency distribution of minimum oxygen saturation in patients who had either laryngeal mask or nasal mask anaesthesia.**Fig. 2.** Histogram showing the frequency distribution of episodes of obstruction in patients who had either laryngeal mask or nasal mask anaesthesia.

The lowest recorded saturations in each group after randomisation are shown in Figure 1. One patient in the LMA group had a saturation of 94% or less as compared to five patients in the nasal mask group (Chi-squared,  $0.05 < p < 0.1$ ). When all the saturation readings in the two groups were compared, mean saturation in the LMA group (98.4% SD 0.91) was significantly better than in the nasal mask group (97.5 SD 3.03, Mann–Whitney *U*-test,  $p < 0.01$ ).

The frequency of episodes of obstruction is shown in Figure 2. The nasal mask group tended to have a higher incidence of multiple episodes of obstruction but this did not reach statistical significance. One patient in the nasal mask group had to be withdrawn from the study; after insertion of the pack at the start of surgery total obstruction occurred on two occasions. He was subsequently managed easily using a LMA. Only one patient in the LMA group had an episode of obstruction not relieved by simple jaw support; in this case the mask became dislodged upwards and forwards during changing of the pack from

**Table 3.** Surgical variables as median (range).

		Group 1	Group 2
Access	Right	3 (1–5)	2 (1–4)
	Left	3 (2–5)	2 (1–3)
Difficulty	Right	3 (2–5)	2 (1–4)
	Left	3 (2–4)	3 (1–5)
Blood	Right	2 (1–3)	2 (1–3)
	Left	2 (1–3)	2 (1–3)

one side to the other and required manipulation to correct the obstruction.

Table 3 shows the surgical variables. There was no significant difference between the groups with respect to access, difficulty of extraction or bleeding, although access tended to be slightly reduced in the LMA group. There was no difference in frequency of arrhythmias or minimum pulse rate between the two groups. Maximum pulse rate in the nasal mask group was significantly higher (nasal 126 SEM 3.01, LMA 117 SEM 3.18,  $p < 0.05$ ).

### Discussion

Previous studies<sup>1,2</sup> have shown that significant numbers of children undergoing dental extractions are subject to episodes of hypoxia. In our study the use of the LMA was associated with fewer episodes of hypoxia than the nasal mask ( $p < 0.1$ ). In addition, the LMA led to a significant increase in mean saturation during the study period ( $p < 0.01$ ). Bone *et al.*<sup>1</sup> found that almost 50% of episodes of hypoxia happened during the recovery period and it would be of interest to determine if the LMA also improved oxygenation during this period.

It would be expected that the better oxygenation with the LMA was associated with a reduced incidence of airway obstruction. Surprisingly, this study failed to demonstrate a significant difference in the observed incidence of airway obstruction between the two groups. Recognition of obstruction with the LMA, especially in conjunction with end-tidal CO<sub>2</sub> monitoring, is much easier and earlier and in almost all cases was easily relieved by gentle upwards pressure on the jaw. This earlier recognition and treatment probably led to the improved saturations because of the lag time in SaO<sub>2</sub> reading.

In order for the LMA to be a suitable alternative in dental anaesthesia it must allow the surgeon adequate access inside the mouth. The results of this study show that although surgical access was somewhat reduced with the LMA this was not statistically significant and did not increase the difficulty of extraction or the amount of bleeding. The increased operating time in the LMA group may reflect the reduced access. Alternatively, the apparent

better airway control may reduce the pressure on the surgeon to complete the extractions as quickly as possible.

The increased maximum pulse rate in the nasal mask group may be a reflection of mild degrees of hypoxia or may be because of lighter levels of anaesthesia associated with the dilution of anaesthetic gas mixtures which almost inevitably occurs as a result of mouth breathing.

In summary, the LMA provides a suitable alternative to the nasal mask as a means of providing anaesthesia for dental extractions in children. Its use is associated with a significant improvement in overall arterial oxygen saturation during the operative period. Airway obstruction is easily recognised and in most cases requires only jaw support to relieve it. The laryngeal mask may be the method of choice where prolonged or difficult extractions are anticipated.

### Acknowledgments

The authors thank the surgical staff of Guy's Dental Hospital for their permission in allowing us to study their patients and for their assistance and patience during this study.

### References

1. BONE ME, GALLER D, FLYNN PJ. Arterial oxygen saturation during general anaesthesia for paediatric dental extractions. *Anaesthesia* 1987; **42**: 879–82.
2. ALLEN NA, ROWBOTHAM DJ, NIMMO WS. Hypoxaemia during outpatient dental anaesthesia. *Anaesthesia* 1989; **44**: 509–11.
3. SAMPAIO MM, CREAN PM, KEILTY SR, BLACK GW. Changes in oxygen saturation during inhalation anaesthesia in children. *British Journal of Anaesthesia* 1988; **61**: 498P.
4. COPLANS MP, CURSON I. Deaths associated with dentistry. *British Dental Journal* 1982; **153**: 357–62.
5. THORNTON JA. General anaesthesia and sedation for dentistry. In: Hewer CL, Atkinson RS, eds. *Recent advances in anaesthesia and analgesia*. 13. Edinburgh: Churchill Livingstone, 1979: 122.
6. BRAIN AIJ. The laryngeal mask—a new concept in airway management. *British Journal of Anaesthesia* 1983; **55**: 801–5.
7. BRODRICK PM, WEBSTER NR, NUNN JF. The laryngeal mask airway. A study of 100 patients during spontaneous respiration. *Anaesthesia* 1989; **44**: 238–241.
8. BEVERIDGE ME. Laryngeal mask anaesthesia for repair of cleft palate. *Anaesthesia* 1989; **44**: 656–7.



## Cardiovascular effects of epidural local anaesthetics

### Comparison of 0.75% bupivacaine and 0.75% ropivacaine, both with adrenaline

H. E. M. KERKKAMP AND M.J.M. GIELEN

#### Summary

*The cardiovascular effects of 20 ml 0.75% bupivacaine with adrenaline 5 µg/ml injected epidurally were compared with those of 20 ml 0.75% ropivacaine with adrenaline. Cardiovascular measurements were performed with a transthoracic electrical bioimpedance monitor. The maximum mean arterial blood pressure decreased significantly from baseline values after both solutions, but the decrease after 20 minutes was more pronounced with bupivacaine (21%) than with ropivacaine (9.6%). Stroke volume increased significantly in both groups (52% for bupivacaine and 29% for ropivacaine). Cardiac output increased significantly from baseline values 2 minutes after epidural administration; the mean of the maximum increase was 64% for bupivacaine and 53% for ropivacaine (NS). The mean of the maximum increase of the ejection fraction was 13% in the bupivacaine group and 9% in the ropivacaine group, but was only significantly different from baseline values following bupivacaine. There was no difference in the onset time or height of the sensory block between the groups. The cardiovascular changes can be ascribed to sympathetic blockade and to systemic absorption of the local anaesthetics and adrenaline.*

#### Key words

*Anaesthetic techniques, regional; epidural.  
Anaesthetics, local; bupivacaine, ropivacaine.*

The cardiotoxic effects of the highly lipid, soluble, local anaesthetic agent, bupivacaine, are well documented,<sup>1,2</sup> and there is a need for an equally effective, but less cardiotoxic agent. Ropivacaine, the propyl homologue of bupivacaine, is a new aminoamide local anaesthetic agent. Unlike bupivacaine, which is a racemic mixture, ropivacaine is prepared as a single enantiomer, S-(-)-1-propyl-2', 6' piperidoxylidide hydrochloride monohydrate.<sup>3</sup> Initial studies in animals and humans suggest that the cardiotoxicity of bupivacaine is greater than that of ropivacaine when given in equal doses on a mg basis.<sup>4-7</sup>

Ropivacaine and bupivacaine, both with adrenaline 5 µg/ml, are equally potent in terms of onset and duration of analgesia after epidural injection,<sup>8</sup> although ropivacaine seems to produce less motor blockade. Cardiovascular changes are comparable with those associated with the autonomic blockade following an epidural.<sup>8,9</sup>

Transthoracic electrical bioimpedance monitoring is a noninvasive method of measuring cardiac output that has been shown to correlate well with cardiac output measured by thermodilution.<sup>10,11</sup> The monitor measures the impedance to electrical current flow from the neck to the base of the thorax. This impedance is a function of the blood

volume in the chest and fluctuates with the cardiac output and the technique is useful in assessing the haemodynamic effects of anaesthetic agents or procedures known to cause cardiovascular changes.<sup>12,13</sup>

The aim of the present study is to determine the cardiovascular changes in relation to epidurally administered bupivacaine and ropivacaine.

#### Methods

As the purpose of the study was to determine the cardiovascular effects of epidural bupivacaine and ropivacaine, the results of any patient who required treatment for hypotension (decrease in systolic arterial pressure > 30%) or bradycardia (heart rate < 50/minute) are not included. The results therefore are from 20 ASA 1 patients, 10 in each group, to which they were randomly allocated. None was receiving any vasoactive medication. All patients were scheduled for elective urological surgery under epidural anaesthesia.

The patients were divided in two groups: group 1 received 20 ml 0.75% ropivacaine with 5 µg/ml adrenaline and group 2 received 20 ml 0.75% bupivacaine with

H.E.M. Kerckamp, MD, Anaesthesiologist, Department of Anaesthesiology, Canisius-Wilhelmina Hospital, Nijmegen, The Netherlands, M.J.M. Gielen, MD, PhD, Associate Professor of Anaesthesiology, Institute for Anaesthesiology, University of Nijmegen, The Netherlands.

Parts of this paper have been presented at the IXth annual ESRA congress, Bern, Switzerland, on 5–8 September 1990.

Accepted 20 June 1990.

Table 1. Demographic data (mean, SEM).

	Ropivacaine 0.75% with adrenaline (n = 10)		Bupivacaine 0.75% with adrenaline (n = 10)		p
Age; years	47	(5)	55	(6)	NS
Height; cm	180	(2)	176	(2)	NS
Weight; kg	79	(4)	75	(3)	NS

n, number of male patients; NS, not significant.

5 µg/ml adrenaline. Premedication consisted of diazepam 10 mg, given orally 90 minutes before inserting the epidural catheter. All patients received 500 ml of a balanced electrolyte solution intravenously before epidural administration of the local anaesthetic agent. A catheter was inserted 3 cm into the epidural space at L<sub>2-3</sub> or L<sub>3-4</sub> and the patient turned supine. They were then allowed to rest quietly for 15 minutes. Systolic, diastolic and mean arterial blood pressures were measured at 1-minute intervals with an automatic monitor. Heart rate, stroke volume, cardiac output and ejection fraction were continuously measured using the NCCOM-3 cardiodynamic monitor (Bomed Medical Manufacturing Ltd., Irvine California USA) for 5 minutes before the epidural injection (baseline) and for 30 minutes after. A test dose of 3 ml of either ropivacaine or bupivacaine both with adrenaline was then administered. After 4 minutes a further 17 ml of the same solution was administered in incremental doses over 4 minutes. Sensory block was determined by pinprick every 5 minutes for 30 minutes.

The haemodynamic measurements are expressed as relative changes from the control values. All variables were compared within and between the two groups. The results were analysed using a *t*-test for paired or unpaired observations. A *p* value less than 0.01 was considered statistically significant.

### Results

Twenty male patients participated in the study. The groups were comparable in age, height and weight (Table 1).

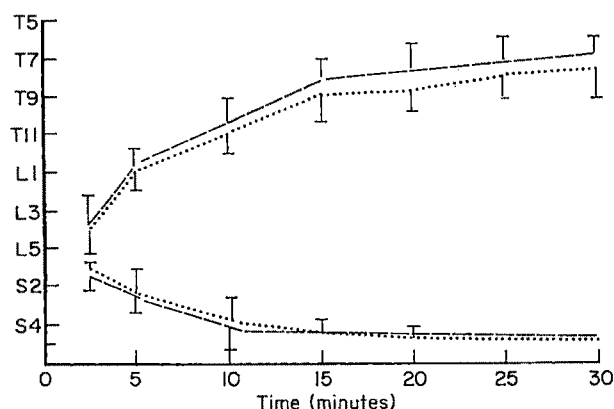


Fig. 1. The onset of sensory blockade after epidural injection of 20 ml of ropivacaine 0.75% (---) and 20 ml of bupivacaine 0.75% (.....) both with adrenaline. Values are means of segmental dermatomes SEM.

Table 2. Mean (SD) pre-blockade haemodynamic values (baseline values).

	Ropivacaine 0.75% with adrenaline (n = 10)	Bupivacaine 0.75% with adrenaline (n = 10)
Heart rate; beats/minute	71 (9)	73 (12)
Systolic arterial blood pressure; mmHg	132 (15)	133 (18)
Diastolic arterial blood pressure; mmHg	80 (10)	83 (9)
Mean arterial blood pressure; mmHg	97 (11)	99 (11)
Stroke volume; ml	97 (24)	79 (24)
Cardiac output; litres/minute	6.8 (1.6)	5.5 (1.5)
Ejection fraction; %	65 (5.7)	60 (6.5)

Table 3. The mean of the maximum changes in percentages.

	Ropivacaine 0.75% with adrenaline	Bupivacaine 0.75% with adrenaline	p
Heart rate	+25	+19	NS
Systolic pressure	-8	-14	NS
Diastolic pressure	-19	-27	NS
Mean pressure	-12	-37	NS
Stroke volume	+29	+52	NS
Cardiac output	+53	+64	NS
Ejection fraction	+9	+13	NS

NS, not significant

**Onset and spread of analgesia.** The development of segmental blockade after the end of epidural administration of the local anaesthetics is shown in Figure 1. There was no significant differences for mean onset time and mean maximum cephalad level of analgesia which reached T<sub>6</sub> for both groups.

**Haemodynamic effects.** The absolute figures of the haemodynamic measurements (baseline values) before injection of the local anaesthetic drug are shown in Table 2. Table 3 shows the mean of the maximum changes after epidural administration. Changes in heart rate are illustrated in Figure 2. Heart rate increased significantly from

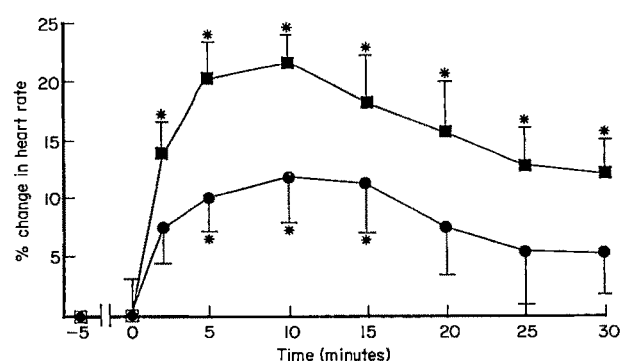


Fig. 2. Changes in heart rate after epidural injection. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural administration. Statistical significant differences within the groups are indicated by (\*). ■—■, ropivacaine; ●—●, bupivacaine.

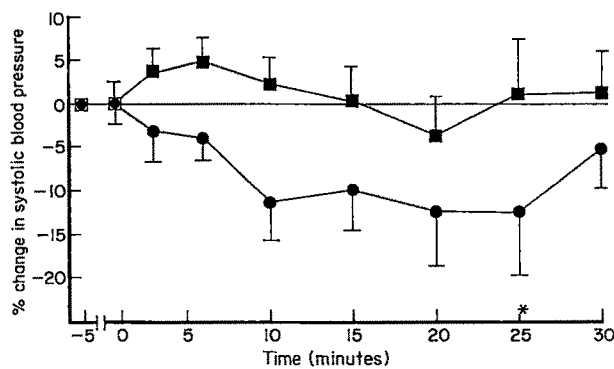


Fig. 3. Changes in systolic blood pressure after epidural injection. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural injections. Significant differences between the two groups are indicated by (\*). ■—■, ropivacaine; ●—●, bupivacaine.

baseline values after 2 minutes in the ropivacaine group and after 5 minutes in the bupivacaine group, but remained significantly elevated for the rest of the study period only in those given ropivacaine. The maximum mean change in heart rate was 22% after ropivacaine and 12% after bupivacaine, after 10 minutes. The changes in heart rate between the two groups were not significantly different.

Changes in systolic blood pressure are shown in Figure 3. The decrease seen was greater in those who had received bupivacaine, but was only significantly different from baseline (−13%) at 25 minutes. In contrast, significant changes in diastolic pressure occurred in both groups compared with baseline, and remained so for the entire study period following bupivacaine and for the first 20 minutes after ropivacaine (Fig. 4). The decrease following bupivacaine was more pronounced than after ropivacaine. Mean arterial blood pressure changes are shown in Figure 5. In the bupivacaine group the mean arterial blood pressure decreased significantly after 2 minutes, but the changes after ropivacaine group were significant only at 20 minutes. The maximum decrease was 12% and 37% for ropivacaine and bupivacaine respectively.

Changes in stroke volume and cardiac output are illustrated in Figures 6 and 7. Stroke volume had increased significantly 2 minutes after bupivacaine and 5 minutes after ropivacaine and in both groups remained significantly

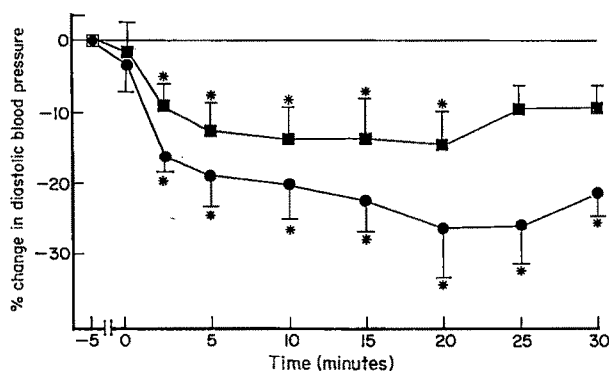


Fig. 4. Changes in diastolic blood pressure. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural administrations. Significant differences within the groups are indicated by (\*). ■—■, ropivacaine; ●—●, bupivacaine.

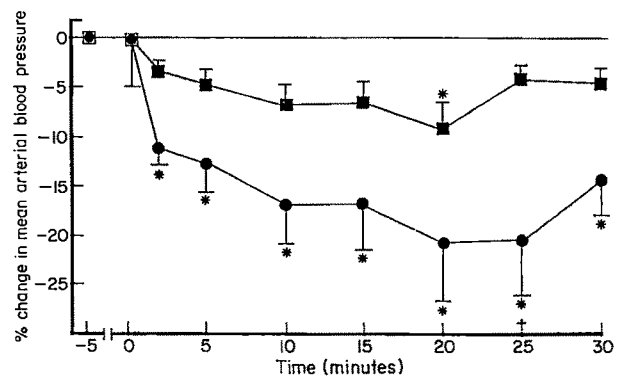


Fig. 5. Changes in mean arterial blood pressure. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural administrations. Significant differences within the groups are indicated by (\*). Significant differences between the two groups are indicated by (†). ■—■, ropivacaine; ●—●, bupivacaine.

raised thereafter. Two minutes after injection, cardiac output had increased significantly in both groups and remained elevated throughout the study period. There were no significant differences between the groups.

The ejection fraction increased significantly in the bupivacaine group at 2 minutes (Fig. 8). The mean of the maximum changes were 9% and 13% for ropivacaine and bupivacaine respectively.

### Discussion

Ropivacaine and bupivacaine are effective local anaesthetic drugs when used for epidural anaesthesia in man; onset times for analgesia were similar in both groups. Other studies have shown a significant differential between sensory and motor blockade with ropivacaine.<sup>8,14</sup>

Cardiovascular effects associated with epidural blockade are related to the type and dose of the local anaesthetic agent, the addition of vasoconstrictors, the level and intensity of the sympathetic blockade and the physiological status of the patient.<sup>15</sup>

In this study, none of the patients were receiving medication which could have influenced physiological status. Because we were only interested in the cardiovascular effects caused by the epidural local anaesthetics, we did not

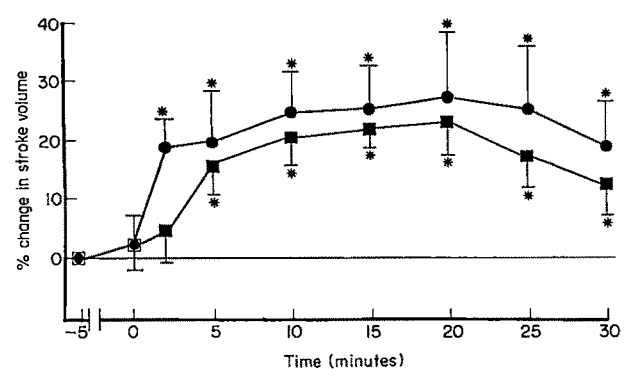


Fig. 6. Changes in stroke volume. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural administrations. Significant differences within the groups are indicated by (\*). ■—■, ropivacaine; ●—●, bupivacaine.

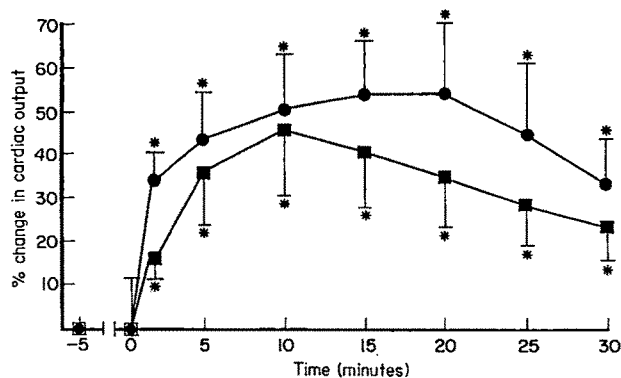


Fig. 7. Changes in cardiac output. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural administrations. Significant differences within the group are indicated by (\*). ■—■, ropivacaine; ●—●, bupivacaine.

include any patient who received vasoactive drugs (ephedrine and atropine) for treatment of hypotension or bradycardia.

The increase in heart rate in both groups is caused by the  $\beta_1$  effects of the absorbed adrenaline. However, this increase was more pronounced in the ropivacaine group. This could be related to the electrophysiological depressant effects of bupivacaine. In an isolated rabbit Purkinje fibre ventricular muscle preparation, ropivacaine was less potent than bupivacaine in terms of its depressant effect on cardiac excitation and conduction.<sup>16</sup> Whether differences exist in the autonomic blockade produced by these two drugs is not known.

The decrease in blood pressure was more pronounced in those who received bupivacaine although not statistically significant. This could be explained either by differences in sympathetic blockade or by different cardiovascular effects of the absorbed local anaesthetics. In this study we did not measure the extent of sympathetic blockade and therefore cannot exclude differences in this aspect.

Ropivacaine has been shown to possess vasoconstrictor properties,<sup>17</sup> and it has also been shown to decrease epidural blood flow by about 37% after epidural administration, in contrast to a 17% increase found following bupivacaine.<sup>18</sup> Animal work has shown that peak plasma levels occur earlier after bupivacaine and ropivacaine.<sup>19</sup> The differences in vasoactivity between the two drugs might be responsible for the differences seen in blood pressure.

Stroke volume and cardiac output increased significantly from control values in both groups; these changes can be ascribed to the absorption of the adrenaline, which was demonstrated in a previous study.<sup>13</sup> There were no significant changes in stroke volume and cardiac output between the two groups.

In a randomised, double-blind study in human volunteers, the effects of intravenous ropivacaine and bupivacaine on left ventricular function were measured.<sup>7</sup> The authors found significant differences in ejection fraction and stroke volume after the end of infusion (mean dose of ropivacaine 150 mg and of bupivacaine 99 mg). The ejection fraction decreased 8% in the ropivacaine group and 10% in the bupivacaine group. In our study, the patients received either 150 mg ropivacaine or 150 mg bupivacaine, both with adrenaline. Ejection fraction increased significantly in the bupivacaine group 2 minutes after epidural

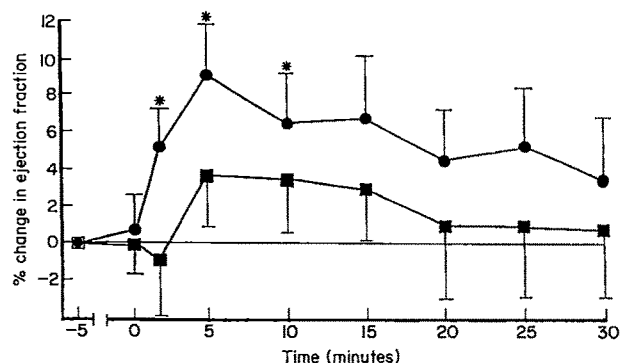


Fig. 8. Changes in ejection fraction. Each point represents the mean (SEM) relative change from control values. T = 0 represents the values at the end of the epidural injections. Significant differences within the bupivacaine group are indicated by (\*). ■—■, ropivacaine; ●—●, bupivacaine.

administration, but there were no significant changes after ropivacaine. The haemodynamic effects of adding adrenaline to local anaesthetics have been shown by several authors,<sup>13, 20-22</sup> and in all these the cardiodepressive effects of the epidurally injected local anaesthetics were reduced or counteracted by the adrenaline. The differences in ejection fraction between the two groups can be caused by the absorbed adrenaline. Less adrenaline would be absorbed in the ropivacaine group than after bupivacaine because of the vasoactive properties of ropivacaine.

The results of the present study suggest that further investigations of ropivacaine and bupivacaine without adrenaline are necessary to determine more accurately the cardiovascular effects after their epidural administration.

## References

- PRENTISS JE. Cardiac arrest following caudal anaesthesia. *Anesthesiology* 1979; **50**: 51-3.
- MALLAMPATI SR, LIU PL, KNAPP RM. Convulsions and ventricular tachycardia from bupivacaine with epinephrine: successful resuscitation. *Anesthesia and Analgesia* 1984; **63**: 856-9.
- ROSENBERG PH, HEINONEN E. Differential sensitivity of A and C nerve fibres to long acting amide local anaesthetics. *British Journal of Anaesthesia* 1983; **55**: 163-7.
- MOLLER RA, COVINO BG. Cardiac electrophysiologic effects of a new long acting local anesthetic agent (LEA-103). *Anesthesiology* 1986; **65**: A183.
- ARTHUR GR, FELDMAN HS, COVINO BG. Comparative pharmacokinetics of bupivacaine and ropivacaine, a new amide local anesthetic. *Anesthesia and Analgesia* 1988; **67**: 1053-8.
- REIZ S, HÄGGMARK S, JOHANSSON G, NATH S. Cardiotoxicity of ropivacaine — a new amide local anaesthetic agent. *Acta Anaesthesiologica Scandinavica* 1989; **33**: 93-8.
- BOWLER GMR, BLOOMFIELD P, FAGAN D, SCOTT DB. The effects of ropivacaine and bupivacaine on the left ventricular function in man. VIII Annual meeting of the European Society of Regional Anaesthesia, Lisboa, Portugal 1989; f.p. 19.
- KERKKAMP HEM, GIELEN MJM, EDSTRÖM HH. Comparison of 0.75% ropivacaine with epinephrine and 0.75% bupivacaine with epinephrine in lumbar epidural anaesthesia. *Regional Anesthesia* 1990; **15**: 204-7.
- FELDMAN HS, ARTHUR GR, COVINO BG. Comparative hemodynamic effects of epidurally administered solutions of ropivacaine and bupivacaine plain and containing epinephrine in the dog. *Anesthesia and Analgesia* 1989; **68**: S84.
- APPEL PL, KRAM HB, MACABEE J, FLEMING AW, SHOEMAKER WC. Comparison of measurements of cardiac output by

- bioimpedance and thermodilution in severely ill surgical patients. *Critical Care Medicine* 1986; **14**: 933-5.
11. BERNSTEIN DP. Continuous noninvasive real-time monitoring of stroke volume and cardiac output by thoracic electrical bioimpedance. *Critical Care Medicine* 1986; **14**: 898-901.
  12. MCMENEMIN IM, KENNY GNC. A computer controlled non-invasive haemodynamic monitoring system. *British Journal of Anaesthesia* 1988; **61**: 492-6.
  13. KERKKAMP HEM, GIELEN MJM. Hemodynamic monitoring in epidural blockade: cardiovascular effects of 20 ml 0.5% bupivacaine with and without epinephrine. *Regional Anesthesia* 1990; **15**: 137-41.
  14. WHITEHEAD E, ARRIGONI B, BANNISTER J. An open study of ropivacaine in extradural anaesthesia. *British Journal of Anaesthesia* 1990; **64**: 67-71.
  15. COVINO BG. Cardiovascular effects of spinal and epidural anesthesia. *Anaesthesist* 1978; **27**: 23-6.
  16. MOLLER RA, COVINO BG. Cardiac electrophysiologic properties of bupivacaine and lidocaine compared with those of ropivacaine, a new amide local anesthetic. *Anesthesiology* 1990; **72**: 322-9.
  17. KOPACZ DJ, CARPENTER RL, MACKEY DC. Ropivacaine vasoconstricts cutaneous blood vessels. *Anesthesiology* 1988; **69**: A 343.
  18. DAHL JB, SIMONSON L, MOGENSEN T, HENRIKSEN JH, KEHLET H. The effect of 0.5% plain ropivacaine on epidural blood flow. *Acta Anaesthesiologica Scandinavica* 1990; **34**: 308-10.
  19. FELDMAN HS, COVINO BG. Comparative motor blocking effects of bupivacaine and ropivacaine, a new amino amide local anaesthetic, in the rat and dog. *Anesthesia and Analgesia* 1988; **67**: 1047-52.
  20. BONICA JJ, AKAMATSU TJ, BERGES PU, MORIKAWA K, KENNEDY WF. Circulatory effects of epidural block: II Effects of epinephrine. *Anesthesiology* 1971; **34**: 514-22.
  21. OTTESEN S, RENCK H, JYNGE P. Haemodynamic alterations secondary to lumbar epidural analgesia and their modification by plasma expansion and adrenaline administration. An experimental study in the sheep. *Acta Anaesthesiologica Scandinavica* 1978; Suppl **69**: 17-31.
  22. STANTON-HICKS M, BERGES PU, BONICA JJ. Circulatory effects of epidural block: IV. Comparison of the effects of epinephrine and phenylephrine. *Anesthesiology* 1973; **39**: 308-14.

## Airway protection by the laryngeal mask

### A barrier to dye placed in the pharynx

R. E. JOHN, S. HILL AND T. J. HUGHES

#### Summary

*Methylene blue was placed in the pharynx of 64 patients undergoing anaesthesia with the laryngeal mask. No leak of dye into the larynx was detected on fiberoptic inspection of the inside of the mask in any subject. The use of the laryngeal mask as a means of protecting the airway during procedures such as minor nasal operations is therefore supported.*

#### Key words

*Anaesthesia, general; instrumentation.*

The laryngeal mask was introduced primarily as a means of airway maintenance.<sup>1</sup> It has already been used during minor ENT procedures, and is reported (clinically) to be safe: without the use of a pack, the airway is protected in the presence of secretions and blood in the pharynx.<sup>2</sup> This study assesses the safety of the technique recommended in the instruction manual.<sup>3</sup>

#### Method

The study was approved by the Ethics Committees of the Royal United Hospital, Bath and the Doncaster Royal Infirmary. Informed consent was obtained from all patients after a full explanation backed up by a patient information sheet. The investigation involved 65 patients whose anaesthetic procedures involved the use of a laryngeal mask: cases that could otherwise be performed with a facemask and oral airway. Anaesthesia was induced with fentanyl 0.5–1.5 µg/kg and propofol 1.5–2.5 mg/kg. The laryngeal mask was inserted and the cuff inflated to achieve a clinically acceptable seal: this was defined as the seal necessary to allow assisted ventilation of the lungs, whether or not a leak was present at maximum inflation. Maintenance of anaesthesia was achieved with 70% nitrous oxide and isoflurane 0.5–2%. The anaesthetic was administered by another anaesthetist and no special instructions were given.

Once spontaneous ventilation had started, 10 ml of 0.1% methylene blue was injected through the mouth into the pharynx. Towards the end of the procedure, the larynx was inspected through the mask using a 4-mm fiberoptic bronchoscope and the presence or absence of blue dye was

noted, in addition to the time the dye had remained in the pharynx before inspection, the position of the mask and any difficulties met in placing it.

The patients were left undisturbed to emerge from anaesthesia and then the mouth and pharynx were suctioned before cuff deflation and removal of the mask. In 10 patients the pharynx was also examined with the bronchoscope passed nasally to ascertain the placement of the dye in the pharynx.

#### Results

One of the patients was excluded from the study because the mask could not be satisfactorily positioned.

Twenty-nine male and 35 female patients were included, with no differences in results between sexes. A size 4 mask was used on all of the male patients and both sizes 3 and 4 in the female patients; difficulties or the presence of a leak were not related to the size of mask used. The time before inspection of the larynx varied from 10 to 70 minutes, with a mean time of 30 minutes. Nasal inspection of the pharynx confirmed that dye had flowed freely into the naso- and upper oro-pharynx: it bathed the cuff of the laryngeal mask.

The results of the study are shown in Table 1. There was a minimal leak of dye in five cases; in these some blue staining was seen around the periphery of the mask. In one other patient, a small amount of dye was seen to pool in the pharynx within the mask, but that was a patient in whom the mask was partially displaced during draping. In no patient was there any staining of the inside of the larynx or

R.E. John, MRCP, FCAaacs, Senior Registrar, Royal Hallamshire Hospital, Sheffield S10 2RX, S. Hill, FFARCS, Consultant, Royal United Hospital, Bath BA1 3QE, T.J. Hughes, FFARCS, Consultant, Doncaster Royal Infirmary, Doncaster DN2 5LT.

Accepted 21 August 1990.



**Table 1.** Results of placement of laryngeal mask.

Leaks	5 (8%)
Positioned first time	57 (87%)
second time	7 (11%)
failed	1 (2%)
Oesophagus seen	6 (9%)
Obstruction caused	4 (6%)

trachea, and no patient coughed or strained during their operation.

Difficulty in inserting the mask was encountered in eight cases, one of whom was the patient not included in the study. On bronchoscopic inspection in that patient, the epiglottis was large and floppy and pushed back against the posterior pharyngeal wall by the mask, preventing further advancement of the mask. In all the other cases, the mask was successfully inserted on the second attempt.

The epiglottis was folded downwards by the mask in eight cases, and in four caused partial obstruction of the airway. The opening of the oesophagus was clearly seen included within the cuff of the mask in six patients.

### Discussion

We questioned the use of the laryngeal mask for cases that involve blood collecting in the pharynx: the results of this study suggest that it does protect the airway. There was no major leak of dye past the mask in any of the cases and no contamination of the airway below the pharynx. Contamination with blood is less likely than with the dye, since it is more viscous and tends to clot. The rapid emergence from anaesthesia performed as described means that the patients can protect their own airways when the mask is removed.

Previous reports of the oesophagus being included within the mask<sup>4</sup> are reinforced; in this study, the incidence was 9%. The mask is not intended to protect patients from aspiration of regurgitated stomach contents and these

results show why it could not reliably do so. The patient with the greatest leak of dye was one in whom the mask moved, backing the practice of securing the mask when used for ENT work.

It should be noted that in one study tracheal tubes with high volume, low pressure cuffs were shown to allow a small leak of dye past folds in the plastic of the cuff in all cases.<sup>5</sup> The only way to avoid leaks altogether involved using red rubber tracheal tubes with low volume cuffs, but these cuffs caused mucosal ischaemia.

Anaesthetising patients who are often day cases for minor procedures on the nose usually involves intubation, with or without the use of a throat-pack and often the use of suxamethonium. The use of the laryngeal mask means that the incidence of sore throat is less,<sup>6,7</sup> that difficult or oesophageal intubations are avoided and that the patients are not exposed to the possible problems of suxamethonium administration.

### Acknowledgements

The ODAs of Bath and Doncaster are thanked for their help and patience.

### References

1. BRAIN AIJ. The laryngeal mask — a new concept in airway management. *British Journal of Anaesthesia* 1983; **55**: 801–5.
2. ALEXANDER CA, LEACH AB. The laryngeal mask — experience of its use in a District General Hospital. *Today's Anaesthetist* 1989; **4**: 200–5.
3. BRAIN AIJ. The Intravent Laryngeal Mask. Instruction manual. 1990.
4. PAYNE J. The use of the fiberoptic laryngoscope to confirm the position of the laryngeal mask. *Anaesthesia* 1989; **44**: 865.
5. SEEGOBIN RD, VAN HASSET GL. Aspiration beyond endotracheal cuffs. *Canadian Anaesthetists' Society Journal* 1986; **33**: 273–9.
6. ALEXANDER CA, LEACH AB. Incidence of sore throats with the laryngeal mask. *Anaesthesia* 1989; **44**: 791.
7. BRODERICK PM, WEBSTER NR, NUNN JF. The laryngeal mask airway. A study of 100 patients during spontaneous breathing. *Anaesthesia* 1989; **44**: 238–41.

## Topical amethocaine in strabismus surgery

D. M. WATSON

### Summary

*A randomised study was performed to assess the effect of topical 1% amethocaine hydrochloride on postoperative analgesia requirements after strabismus surgery. Forty children scheduled for elective operation were allocated randomly to receive either topical amethocaine or normal saline. Postoperative analgesia was evaluated with the use of a four-point assessment score and analgesic requirements. The topical amethocaine provided significantly better postoperative analgesia ( $p < 0.001$ ) as measured by both the assessment score and the postoperative analgesia requirement.*

### Key words

*Anaesthetics, topical; amethocaine.*

*Surgery; strabismus.*

The problems of anaesthesia for surgery to correct strabismus in children include bradycardia, nausea and vomiting and postoperative pain relief. A common anaesthetic technique combines the use of atropine to obtund the oculocardiac reflex and intramuscular opioids to provide postoperative analgesia.

Diamond described the use of topical anaesthesia alone in adult patients undergoing strabismus surgery and suggested that this technique was beneficial since it avoided the potential hazards of general or retrobulbar anaesthesia.<sup>1</sup> The major disadvantage was the incidence of postoperative patient discomfort.

Topical amethocaine is used routinely to provide conjunctival anaesthesia in casualty and ophthalmology departments to enable full examination of the eye. The aim of this study was to assess the effect of conjunctival anaesthesia provided by topical 1% amethocaine hydrochloride on the postoperative analgesia requirements after strabismus surgery in children.

### Methods

Following Ethics Committee approval informed consent was obtained from the parents of 40 children aged 1–12 years (mean 4 years) presenting for elective surgery for correction of strabismus. Children weighing less than 10 kg or those presenting for repeat surgery were not studied. All patients were in ASA group 1.

The patients were allocated randomly to two groups. Randomisation instructions were placed in sealed

envelopes at the beginning of the trial, which were then mixed and numbered and placed in the anaesthetic room of the ophthalmic operating theatre. When the patient arrived in the anaesthetic room the anaesthetist in charge of the case opened the next envelope and followed the randomisation instructions enclosed. Both groups received a standard premedication 90 minutes before operation consisting of trimeprazine 3 mg/kg and EMLA cream to the proposed venepuncture site. Immediately before the induction of anaesthesia each patient received intravenous atropine 0.02 mg/kg. Anaesthesia was induced with thiopentone 4 mg/kg and the trachea was intubated following suxamethonium 1 mg/kg. Anaesthesia was maintained with the patient breathing spontaneously nitrous oxide 66% and halothane 1–3% in oxygen. During the procedure the patients were monitored using ECG, pulse oximetry, noninvasive blood pressure monitoring and a temperature probe. Postoperative analgesia was prescribed on an 'as required basis' and consisted of paracetamol suspension orally (age 1–5 years 120–250 mg; 6–12 years 250–500 mg) or intramuscular pethidine 1 mg/kg.

Following intubation of the trachea and again immediately before extubation, the trial group had two drops of 1% amethocaine hydrochloride put into the eye being corrected. Normal saline was used at the same times in the control group. The times of the second administration and extubation were recorded.

The patients were assessed for pain on arrival in the recovery ward and at 15 and 30 minutes, and at 1, 2, 4, 6 and 8 hours after operation. The assessor (D.W.) was blind

D.M. Watson, MB, ChB, FFARCSI, Senior Registrar, University Department of Anaesthesia, Medical School, Beech Hill Road, Sheffield S10 2RX.

Accepted 19 June 1990.

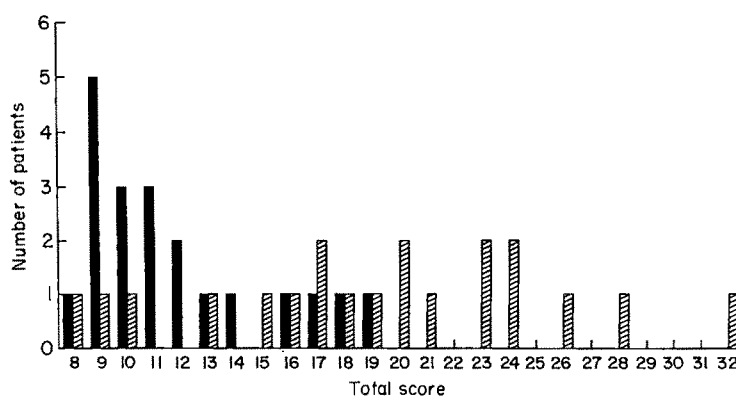


Fig. 1. Total postoperative assessment scores; ■, trial group; ▨, control group.

to the anaesthetic technique used. The children were transferred back to the ward after 30 minutes. Parents were encouraged to be present in the anaesthetic room and when their child returned to the ward.

Assessment was made by the author using a four-point scale: 1, sleeping; 2, awake and quiet; 3, agitated; 4, crying. The pulse and respiratory rate were noted and the times of administration of any analgesics recorded.

Statistical analysis was undertaken using the Chi-squared test for analgesic requirements and the Kruskal-Wallis one-way nonparametric test for assessment scores.

### Results

The children varied in age from 1–12 years (mean 4 years) and their weight from 11–46 kg (mean 20.6 kg). The length of the procedure varied from 13 to 50 minutes (mean 26 minutes).

Fifteen out of 20 patients (75%) in the trial group required no further analgesia. The remaining five patients (25%) received oral paracetamol suspension, which was given at between 1 and 4 hours after operation. No patients in this group required pethidine. In the control group only three patients (15%) received no further analgesia; six (30%) received paracetamol at between 30 minutes and 2 hours after operation; six (30%) received pethidine at between 30 minutes and 2 hours after operation and five (25%) received both paracetamol and pethidine between 30 minutes and 2 hours after operation. There was a significant difference between the two groups ( $p < 0.001$ ).

The total assessment scores varied from 8 to 32. The mean score for the trial group was 11.4 (range 8–19) and for the control group 19.5 (range 8–32) (Fig. 1). There was a significant difference between the two groups ( $p < 0.001$ ).

Only three (7.5%) of the 40 patients in the trial had any nausea or vomiting. One was in the control group and two were in the trial group.

### Discussion

Infiltration of amethocaine into the extra-ocular muscles has no analgesic effect and therefore postoperative pain following strabismus surgery would seem to be conjunctival.

Diamond<sup>1</sup> describes the use of topical anaesthesia in strabismus surgery to provide analgesia both during and

after operation. The findings of this study show that topical 1% amethocaine hydrochloride gives significant postoperative analgesia and results in a significant decrease in the postoperative requirements of both paracetamol and pethidine.

The assessment of postoperative pain in children is more difficult than in adults because of their relative inability to communicate, fear of the hospital surroundings and the difficulty that they may have in differentiating pain from nausea or other subjective sensations. In this trial parents were encouraged to be present in the anaesthetic room and as soon as their child returned to the ward, in an effort to decrease the distress caused by fear of the hospital environment.

Visual analogue toys<sup>2</sup> have been used to assess pain in children and in those aged over 4 years they appear to achieve this. It was not possible to use toys in this study because many of the children were less than 4 years old. The four-point assessment score was used instead and gave a subjective measure of postoperative pain; objective measurement was obtained from the requirement for postoperative analgesics.

Strabismus surgery is associated with incidences of vomiting that range from 50 to 85%.<sup>3–5</sup> The existence of an oculo-emetic reflex has been suggested.<sup>4–5</sup> The 7.5% incidence of vomiting in this study supports the suggestion that pre-operative anticholinergics should be used to reduce the morbidity of vomiting after strabismus surgery.

In conclusion, it is suggested that topical 1% amethocaine hydrochloride may be used routinely in anaesthesia for strabismus surgery since it provides significant postoperative analgesia and decreases the requirement for intramuscular opiates in the postoperative period.

### Acknowledgments

I thank Dr B.R. Milne, Consultant Anaesthetist, Doncaster Royal Infirmary, for his support and help during this study and Mr L.R. Kolli, Consultant Ophthalmic surgeon, Doncaster Royal Infirmary, for his permission to include his patients in the study.

### References

1. DIAMOND GR. Topical anaesthesia for strabismus surgery. *Journal of Pediatric Ophthalmology and Strabismus* 1989; **26**: 86–90.

2. WHITE JB, STOW P. Rationale and experience with visual analogue toys. *Anaesthesia* 1985; **40**: 601-3.
3. HADAWAY EG, INGRAM RM, TRAYNAR MJ. Day case surgery for strabismus in children. *Transactions of the Ophthalmological Societies of the United Kingdom* 1977; **97**: 23-5.
4. VAN DEN BERG AA, LAMBOURNE A, CLYBURN PA. The oculoemetic reflex. *Anaesthesia* 1989; **44**: 110-7.
5. MCGOLDERICK KE. Paediatric anaesthesia for ophthalmic surgery. In: *Anaesthesia for ophthalmology*. Alabama: Aesculapius Publishing, 1982: 51-83.

## Caesarean section in undiagnosed Eisenmenger's syndrome

Report of a patient with a fatal outcome

D. H. GILMAN

### Summary

*An obstetric patient is described in whom the first sign of cardiac disease was unexplained hypoxaemia during emergency anaesthesia for antepartum haemorrhage, with an eventual fatal outcome. The case highlights the importance of patient information at the booking clinic, and the implications of a raised haemoglobin in early pregnancy.*

### Key words

*Anaesthesia; obstetric.*

*Complications; Eisenmenger's syndrome.*

Cardiac disease remains an important cause of indirect maternal mortality,<sup>1</sup> which is defined as mortality from the physiological changes of pregnancy aggravating previous existing disease, or disease developing in pregnancy but unrelated to the pregnancy. It accounted for 17 deaths in the three years 1982–1984, of which six were related to congenital heart disease and five of these were Eisenmenger's complex. The latter was originally described in 1897<sup>2</sup> in a 32-year-old male who collapsed and died following an haemoptysis. There had been a childhood history of dyspnoea and cyanosis and the postmortem findings were of a large ventricular septal defect, large right ventricle and atherosclerosis in the pulmonary vessels. In 1968, Wood<sup>3</sup> delineated the syndrome by stating that pulmonary hypertension was an essential feature and could result from a variety of cardiac causes, but all ultimately led to a reversible or bidirectional shunt.

These patients are particularly at risk in the postpartum period because of the haemodynamic changes of labour and the puerperium.

### Case history

A 31-year-old Caucasian female was first seen in the booking clinic at 11 weeks' gestation. Her previous medical history consisted of a heart murmur detected as a child but for which no treatment had been given. She described her health as good, but admitted to habitual moderate smoking until becoming pregnant. Examination at booking revealed a well-looking obese woman, 153 cm tall and weighing 76 kg. Examination of the cardiovascular system showed

her to be in sinus rhythm with an arterial blood pressure of 100/80 mmHg. No murmurs or abnormal heart sounds were detected. Her physical examination was unremarkable, but a raised haemoglobin level of 16.3 g/dlitre was unfortunately overlooked.

She presented at 36 weeks' gestation with a 3-hour history of vaginal bleeding. On examination she appeared well, with a tense, tender uterus of 36 weeks' size. Immediate ultrasound examination showed there to be a single, viable fetus and a grade I placenta praevia. It was decided to perform a Caesarean section.

She arrived in the operating theatre extremely anxious with a blood pressure of 160/90 mmHg. Induction followed pre-oxygenation, with the patient in left lateral tilt, using thiopentone 300 mg, suxamethonium 100 mg, nitrous oxide, oxygen and isoflurane. Atracurium was given following recovery from suxamethonium. Following delivery of a live male infant a total of 10 mg of diamorphine was given intravenously to minimise use of isoflurane. The cardiovascular system remained stable until after 45 minutes, when the patient's colour was noticed to have deteriorated and simultaneously the obstetrician reported the ease with which tissues bruised. Clotting studies revealed a picture of early disseminated intravenous coagulation with a platelet count of  $66 \times 10^9$ /litre and kaolin cephalin time (KCT) ratio of 57/42. Estimated blood loss was 500 ml.

The patient remained haemodynamically stable over the next 10 hours, but developed petechiae haemorrhages around the wound. The uterus, initially well contracted, became enlarged and soft and its contents were evacuated

D.H. Gilman, BSc, FFARCS, Senior Registrar, Department of Anaesthetics, King's College Hospital, Denmark Hill, London SE5 9RS.

Accepted 14 December 1989.

**Table 1.** Decline in arterial oxygenation over the 72 hours in the intensive care unit, and the expected  $PaO_2$  assuming no shunt.

	Admission to ITU	Hours				
		+6	+12	+24	+48	+72
$FiO_2$	80%	60%	50%	21%	40%	50%
pH	7.33	7.34	7.34	7.44	7.44	7.44
$PO_2$ kPa	15.4	10.9	6.5	5.8	4.9	3.5
$PCO_2$ kPa	4.45	5.4	5.3	4.7	4.5	4.4
$HCO_3$ mmol/litre	19.6	21.8	21.9	24.0	23.1	22.4
Saturation	98%	94%	82%	79%	71%	51%
Mode of ventilation	IPPV	IPPV	CPAP	Spontaneous	Spontaneous	Spontaneous
Expected $PaO_2$ , kPa	70	52	42	12	32	42

under general anaesthesia when the circulating blood volume had become restored with blood and cryoprecipitate. This was a protracted procedure of 2.5 hours with brisk uterine bleeding that necessitated packing the uterine cavity and re-opening the abdominal wound. Total estimated blood loss was 1500 ml and cyanosis and a coagulopathy were obviously present. Mechanical and supply causes of a decreased oxygen content were excluded.

The patient's poor and deteriorating condition warranted transfer to the intensive care unit for further management and investigation. Regrettably, no measurements of arterial oxygenation was made after Caesarean section and before evacuation of the retained products. An electrocardiogram revealed sinus rhythm, with marked right axis deviation and right ventricular hypertrophy with strain. Chest X-ray showed the heart to be normal size with prominent pulmonary arteries and oligemic lung fields, which suggested right to left shunt. This was further suggested by failure of arterial oxygen saturation to improve despite repeatedly increasing the inspired oxygen tension. The diagnosis of Eisenmenger's syndrome was now entertained.

Her condition improved in the following 48 hours and she was weaned from mechanical ventilation and her trachea extubated. She received further blood, fresh frozen plasma and platelet infusions, but improvement was not sustained and she developed further respiratory distress with tachypnoea and hypoxaemia, but refused further ventilatory support. The blood gases during this time are shown in Table 1.

Arrangements had been made meanwhile for transfer to a cardiothoracic unit. She suffered an hypoxic cardiac arrest soon after, from which resuscitation was not possible. Postmortem findings were a persistent ductus arteriosus, large thick-walled right ventricle, and pulmonary vascular changes of hypertension with atheroma.

### Discussion

The unusual feature of this case was the absence of cardiac or respiratory symptoms both before and during pregnancy. Only the raised haemoglobin, in a patient known to be a smoker, was noteworthy at the time of booking when physical examination had been unremarkable. The lack of clinical signs and symptoms in a patient who had developed Eisenmenger's syndrome may be striking. In a similar report, cyanosis in the absence of either a heart murmur or finger clubbing first became apparent 3 months after delivery.<sup>4</sup> Presumably, if the pulmonary and systemic

pressures have equalised, no turbulent blood flow occurs.

The optimum method of anaesthesia in Eisenmenger's syndrome remains controversial. On theoretical grounds a decrease in systemic vascular resistance would promote right to left shunting, yet regional anaesthesia seems to be well tolerated.<sup>5</sup> However, in a recent report, epidural anaesthesia was administered after insertion of a balloon-tipped pulmonary artery flotation catheter and invasive arterial monitoring, but death occurred on the sixth day after operation from cerebral thromboembolism.<sup>6</sup> There were no signs of shunt reversal during the epidural anaesthetic.

To avoid the risk of increased right to left shunt, the use of a vasoconstrictor before induction of general anaesthesia has been reported by Bird and Strunin.<sup>7</sup> They, however, deprecated the use of such a technique as a routine, since it resulted in systemic hypertension and further oxygen desaturation.

It appears that both regional and general anaesthesia are well tolerated in this condition,<sup>5,8</sup> but life-threatening complications are likely to occur in the postpartum period. Their incidence and severity depend upon the nature of the underlying cardiac defect.<sup>4</sup> Hypoxaemia, thrombo-embolism and cardiac arrhythmias are most often implicated. There is a complex relationship between pulmonary vascular resistance, acidosis and hypoxia; these latter two independently increase pulmonary vascular resistance, but the superimposition of acidosis upon hypoxaemia potentiates this response.<sup>9</sup> It may be that avoidance of these factors with careful attention to reduction and replacement of blood loss and maintenance of acid-base balance are more important than the choice of anaesthetic technique.

In conclusion, an abnormal haemoglobin result, whether above or below the range of the physiological anaemia of pregnancy requires careful evaluation, especially if the patient gives any history of childhood cardiac disease. Non-invasive investigations such as echocardiography and ECG may be safely applied in pregnancy to help with diagnosis. Anaesthetists, when called upon to provide emergency obstetric anaesthesia, should be aware of the possibility of severe cardiac disease when there is an apparently isolated raised haemoglobin in pregnancy and ensure appropriate monitoring is used. Pulse oximetry would be particularly useful, since it would indicate the balance between the pulmonary and systemic circulations.

### Acknowledgments

I thank Dr S. Brookes, Registrar, Department of Anaesthetics, and Mr B. Hackman, Consultant Obstetrician, for their permission to publish this case



report, and Dr C. E. Blogg and Dr H. Smith, Consultant Anaesthetists for their help and encouragement.

### References

1. Report on Confidential Enquiries into Maternal Deaths in England and Wales 1982-1984. HMSO No. 34.
2. EISENMENGER V. Die Angeborenen Defecte der Kammschiedewand des Herzens. *Zeitschrift für Klinische Medizin* 1897; **32**: (Suppl.) 1-28.
3. WOOD P. The Eisenmenger syndrome or pulmonary hypertension with reversed central shunt. *British Medical Journal* 1958; **2**: 701-9, 755-62.
4. GLEICHER N, MIDWALL J, HOCHBERGER D, JAFFIN H. Eisenmenger's syndrome and pregnancy. *Obstetrical and Gynecological Survey* 1979; **34**: 721-41.
5. SPINNATO JA, KRAYNACK BJ, COOPER MW. Eisenmenger's syndrome in pregnancy: epidural anesthesia for elective Cesarean section. *New England Journal of Medicine* 1981; **304**: 1215-7.
6. HYTENS L, ALEXANDER JP. Maternal and neonatal death associated with Eisenmenger's syndrome. *Acta Anaesthesiologica Belgica* 1986; **37**: 45-51.
7. BIRD TM, STRUNIN L. Anaesthesia for a patient with Down's syndrome and Eisenmenger's complex. *Anaesthesia* 1984; **39**: 45-50.
8. LUMLEY J, WHITWAM JG, MORGAN M. General anaesthesia in the presence of Eisenmenger's syndrome. *Anesthesia and Analgesia* 1977; **56**: 543-47.
9. RUDOLPH AM, YUAN S. Response of the pulmonary vasculature circulation to hypoxia and H<sup>+</sup> ion concentration changes. *Journal of Clinical Investigation* 1966; **45**: 399-405.

## Hypermetabolism in arthrogryposis multiplex congenita

P. M. HOPKINS, F. R. ELLIS AND P. J. HALSALL

### Summary

*Two patients who developed hypermetabolic reactions during anaesthesia and surgery and who were suffering from arthrogryposis multiplex congenita are reported and it is proposed that the reaction is distinct from malignant hyperthermia and independent of the anaesthetic agents used. The implications for anaesthetists involved in the management of patients with arthrogryposis multiplex congenita are discussed.*

### Key words

*Complications; arthrogryposis multiplex congenita. Hyperthermia.*

Anaesthesia in patients with arthrogryposis multiplex congenita (AMC) is sparsely documented in the literature. However, in six out of nine reports of anaesthesia in this condition<sup>1–9</sup> an increase in body temperature was recorded. Furthermore, in five of these case reports<sup>3,4,6–8</sup> the pyrexial response was assumed to indicate that the patient was susceptible to malignant hyperthermia (MH), without the benefits of recognised procedures for the diagnosis of MH susceptibility<sup>10</sup> which have since been published. This has led at least one standard text of anaesthesia<sup>11</sup> to associate AMC with MH.

We believe the hypermetabolic response observed during anaesthesia and surgery in these patients is distinct from MH because of our experience with two families with children with AMC.

### Case histories

#### Case 1

A 2-year-old boy with AMC was anaesthetised for correction of multiple web syndrome. Following induction with cyclopropane, halothane and suxamethonium his trachea proved difficult to intubate; his temperature at this time was 37.8°C. After 40 minutes the temperature had risen to 38.7°C and biochemical investigation showed a plasma potassium level of 5.7 mmol/litre and pH of 7.125. Anaesthesia was discontinued because the child, with pyrexia, acidosis and hyperkalaemia, was assumed to have MH. Treatment with bicarbonate, dantrolene and body-

cooling returned the body temperature and biochemical indices to within normal limits after one hour and the child appeared well.

He was thought to be too young to submit for muscle biopsy, so both parents were subsequently investigated for MH susceptibility by *in vitro* muscle contracture testing with halothane and caffeine according to the protocol of the European Malignant Hyperthermia Group<sup>10</sup> and were found to be normal.

#### Case 2

A 5-year-old Afghan boy with AMC was to undergo release of contractures of the fingers of his right hand and tendon transfers. He was premedicated with trimeprazine 3 mg/kg and anaesthetised with thiopentone, fentanyl and atracurium. Tracheal intubation was achieved easily, intermittent positive pressure ventilation started and anaesthesia maintained with enflurane and nitrous oxide in oxygen. Monitoring included ECG, blood pressure, nasopharyngeal temperature and end-tidal carbon dioxide measurement. After 45 minutes the end-tidal carbon dioxide began to rise, the heart rate increased from 110 to 140 and the temperature began rising at a rate of 0.1°C every 5 minutes. Active cooling was started when the temperature reached 38°C, the enflurane stopped and anaesthesia continued with nitrous oxide in oxygen and increments of fentanyl. This regimen successfully resolved the hypermetabolic response and the procedure was completed without further incident. However, a tempera-

P.M. Hopkins, MB, BS, FCAnaes, Lecturer, F.R. Ellis, PhD, FCAnaes, Reader, P.J. Halsall, MB, ChB, Clinical Assistant, University Department of Anaesthesia, Clinical Sciences Building, St James's University Hospital, Leeds LS9 7TF.

Accepted 21 August 1990.

ture of 38°C developed 2 hours after operation which also responded to cooling. Biochemical studies at the time of the perioperative pyrexia showed normal serum potassium, creatine kinase and pH values. The first specimen of urine passed after operation contained no myoglobin.

Five months later the surgical procedure was repeated on the left hand. Anaesthesia was induced with thiopentone, and maintained with pancuronium and infusions of fentanyl and propofol as well as nitrous oxide in oxygen. Despite the use of agents which are known not to trigger MH an hypermetabolic response also occurred during this procedure. After one hour of anaesthesia, there was a rise in body temperature, tachycardia and increased end-tidal carbon dioxide. These changes, which were reversed by active cooling measures, recurred when the cooling was stopped and controlled once more by cooling which was continued for the remainder of the procedure.

### Discussion

MH has been associated with several conditions, mostly musculoskeletal abnormalities, but these associations have been shown to be probably coincidental other than that with central core disease.<sup>12</sup>

If the incidence of MH in AMC is as high as might be inferred from the cases of AMC reported in the literature,<sup>1-9</sup> patients with AMC and their families would have to be assumed to be MH susceptible until their MH status had been diagnosed by muscle biopsy and *in vitro* contracture testing.

In their review of anaesthetic experience in 67 patients with AMC, Baines *et al.*<sup>9</sup> reported that none suffered any untoward event. However, the authors admit that temperature was not always recorded. It is therefore possible that hypermetabolic responses went undetected. The tendency for an individual child with AMC to develop an hypermetabolic response may depend on whether the aetiology of the arthrogryposis is primarily neurogenic or myogenic and this may be one of the reasons why this type of reaction is not always seen.

The first patient we have reported was assumed to be an MH reaction, but subsequent parental investigations showed that this was not the case, unless the child had exhibited a spontaneous mutation, which is thought to be unlikely. It might be argued that without performing muscle biopsies and *in vitro* contracture testing on the parents of the second child it is impossible to exclude the child from being MH susceptible. However, we believe there are sufficient criteria to conclude that the hypermetabolic response was not MH: the normal results of the biochemical investigations carried out at the time of the first reaction; the successful treatment of each reaction with cooling measures and most significantly, the occurrence of

an hypermetabolic response in the absence of MH triggering drugs.

It is important that staff anaesthetising a patient with AMC are aware that there is a possibility of an hypermetabolic response that is distinct from MH, because the AMC response will respond to active cooling, while MH requires the cessation of triggering agents, cooling, intravenous dantrolene, the abandonment of the surgical procedure and intensive therapy of any ensuing metabolic derangement.

We conclude, therefore, that an hypermetabolic response to anaesthesia and surgery occurs in patients with AMC that is distinct from MH, and that this response is independent of the type of anaesthetic agents used. This type of response should be anticipated, appropriate monitoring for its detection used, and methods of cooling available.

Finally, it should be remembered that it is possible for two rare conditions, such as AMC and MH, to be present coincidentally in the same patient.

### References

1. OBEROI GS, KAUL HL, GILL IS, BATRA RK. Anaesthesia in arthrogryposis multiplex congenita. Case report. *Canadian Journal of Anaesthesia* 1987; **34**: 288-90.
2. YODONO M, TANIGUCHI K, MATSUKI A, OYAMA T. Anesthesia for a patient with congenital arthrogryposis. *Masui Japanese Journal of Anesthesiology* 1983; **32**: 871-5.
3. HONDA N, KONNO K, ITOHDA Y, NISHINO M, MATSUSHIMA S, HASEBA S, HONDA Y, GOTOH Y. Malignant hyperthermia and Althesin. *Canadian Anaesthetists Society Journal* 1977; **24**: 514-21.
4. ITOHDA Y, ODA S, NISHINO M, IMANO K, HASEBA J. Anaesthetic experience in congenital arthrogryposis multiplex. *Masui Japanese Journal of Anesthesiology* 1976; **25**: 697-703.
5. CONRAD JA, FROST HM. Evaluation of subcutaneous heel-cord lengthening. *Clinical Orthopedics* 1969; **64**: 121-7.
6. RELTON JES, CREIGHTON RE, JOHNSTON AE, PELTON DA, CONN AW. Hyperpyrexia in association with general anaesthesia in children. *Canadian Anaesthetists Society Journal* 1966; **13**: 419-24.
7. KING JO, DENBOROUGH MA, ZAPF PW. The myopathies of malignant hyperpyrexia. *Clinical Studies in Myology, International Congress Series No. 295*, 535-42.
8. BAUDENDISTEL L, GOUDSOUZIAN N, COTE C, STRAFFORD M. End-tidal CO<sub>2</sub> monitoring. Its use in the diagnosis and management of malignant hyperthermia. *Anaesthesia* 1984; **39**: 1000-3.
9. BAINES DB, DOUGLAS ID, OVERTON JH. Anaesthesia for patients with arthrogryposis multiplex congenita: what is the risk of malignant hyperthermia? *Anaesthesia and Intensive Care* 1986; **14**: 370-2.
10. EUROPEAN MALIGNANT HYPERTHERMIA GROUP. A protocol for the investigation of malignant hyperthermia (MH) susceptibility. *British Journal of Anaesthesia* 1984; **56**: 1267-9.
11. ATKINSON RS, RUSHMAN GB, LEE JA. *A synopsis of Anaesthesia*, 10th edn. Bristol: John Wright, 1987.
12. BROWNELL AKW. Malignant hyperthermia: relationship to other diseases. *British Journal of Anaesthesia* 1988; **60**: 303-8.

## Chickenpox pneumonia, its complications and management

A report of three cases, including the use of extracorporeal membrane oxygenation

G. P. M. CLARK, P. M. DOBSON, A. THICKETT AND N. M. TURNER

### Summary

*We report three cases of chickenpox pneumonia in adults, all of whom required intermittent positive pressure ventilation. One patient developed a variety of complications, and another, a pregnant woman, required extracorporeal membrane oxygenation.*

### Key words

*Infection; chickenpox.*

*Complications; pneumonia.*

*Lungs; extracorporeal membrane oxygenation.*

Chickenpox (varicella) is a highly infectious disease, caused by the varicella-zoster virus. Few people escape the disease as children, hence its relative rarity in adults. However, whilst the disease usually runs a relatively benign course in healthy children, pneumonia is a common complication of varicella infection in adults, with a reported incidence of 16–50%.<sup>1,2</sup> Varicella pneumonia is associated with a significant morbidity and mortality. We report three cases presenting to our unit within a period of 3 months. All three patients recovered, and each presented different features of the disease. The first patient had a relatively straightforward course; the second developed a variety of complications, including myocardial infarction, left hemiplegia and disordered liver function; the third required extracorporeal membrane oxygenation (ECMO) to maintain adequate oxygenation.

### Case histories

#### Case 1

A 54-year-old miner was admitted to the infectious diseases unit with a 2-day history of a widespread rash, typical of chickenpox. His granddaughter had developed chickenpox 2 weeks previously. On admission, there were no respiratory symptoms and his chest was clear. Past medical history revealed little of note, other than that he was a heavy smoker.

The following day he became increasingly dyspnoeic and a chest X ray showed patchy consolidation of both

lung fields. Varicella pneumonia was diagnosed, and treatment with acyclovir 10 mg/kg three times daily was started. Cefuroxime was also given as prophylaxis against bacterial infection. He was then transferred to the intensive care unit (ICU) at this hospital. On admission, he had a respiratory rate of 50 he was unable to talk, and blood gas analysis revealed  $P_{aO_2}$  of 6.5 kPa on an inspired oxygen of 60%. His trachea was intubated, and ventilation was controlled artificially. Initially he required an inspired oxygen of 90%, with a positive end-expiratory pressure (PEEP) of +5.5 mmHg to maintain a  $P_{aO_2}$  of 10 kPa. His respiratory function gradually improved and artificial ventilation was discontinued after 24 days. He was discharged home after 39 days in hospital. He was asymptomatic when seen 6 months later; his chest X ray was normal, and respiratory function tests showed only a mild reduction in transfer factor.

#### Case 2

A 39-year-old man, also a miner and a heavy smoker, was admitted to the infectious diseases unit having caught chickenpox from his daughter. He had a 4-day history of rash and a 2-day history of increasing shortness of breath and pleuritic chest pain. His chest X ray showed features compatible with varicella pneumonia (Fig. 1). Treatment was started with acyclovir 10 mg/kg three times daily and cefuroxime, and he was transferred to our unit for observation.

G.P.M. Clark, MB, BChir, MRCP, FFARCS, Consultant, P.M. Dobson, MB, BS, FCAnaes, A. Thickett, MB, BS, FCAnaes, N.M. Turner, MB, BS, FCAnaes, Registrars, Department of Anaesthesia, Northern General Hospital, Herries Road, Sheffield S5 7AU.

Accepted 4 August 1990.

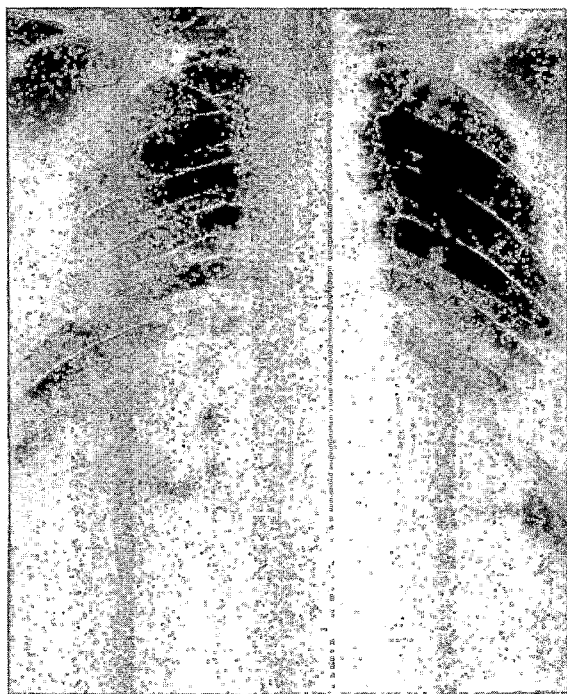


Fig. 1. Chest X ray of Case 2, showing patchy shadowing of both lung fields, most marked at the bases, features compatible with chickenpox pneumonia.

On admission to the ICU, his arterial  $P_{aO_2}$  was 8.3 kPa on an inspired oxygen of 40%. He remained stable for the next 48 hours, but needed an inspired oxygen of 50% to maintain an adequate oxygen saturation. On the third day, he deteriorated suddenly, with a decrease in  $P_{aO_2}$  to 5.2 kPa, and he required tracheal intubation and artificial ventilation. An initial improvement was followed one hour later by a sudden deterioration. Oxygen saturation decreased to below 80%, and his systolic blood pressure decreased from 120 to 70 mmHg. The electrocardiogram (ECG) showed a variety of arrhythmias, including first degree heart block and Mobitz type 1 and type 2 block. A 12-lead ECG showed the changes of acute inferior myocardial infarction. This diagnosis was confirmed by cardiac enzyme studies.

A flow-directed balloon-tipped pulmonary artery catheter was introduced, and by giving a combination of dobutamine and sodium nitroprusside, cardiac output and oxygen delivery were improved. The  $P_{aO_2}$  was maintained at 10–11 kPa with an inspired oxygen of 70%. The patient gradually improved, and after 7 days the  $P_{aO_2}$  was 13.5 kPa on an inspired oxygen of 50%.

On the fourth day, he became jaundiced and his plasma transaminases and alkaline phosphatase were increased markedly. A liver ultrasound was reported as being 'slightly less echogenic than expected, possibly representing inflammation. Gall bladder and biliary tree were normal.' Liver function tests returned to normal after 14 days.

On day 12, his temperature increased to 39°C. This pyrexia continued for the next 19 days, was non-fluctuant, and was associated throughout this period with a normal white cell count. No cause was found, despite extensive bacterial cultures and repeated changes of venous catheters. On two occasions *Enterobacter cloacae*, sensitive to cefotaxime, was isolated from the sputum. Treatment with this

antibiotic, and subsequently with a variety of others given empirically, made no difference to his temperature. It was suggested therefore that this could have been a manifestation of chickenpox encephalitis, affecting his temperature-regulating mechanism.

On day 19, he was not moving his left arm or leg and he had a left extensor plantar response. When artificial ventilation was discontinued on day 26, and all sedative drugs were withdrawn, a full neurological examination revealed a left hemiparesis, decreased comprehension, and a left palatal and vocal cord paresis. He could not swallow food and had an incompetent larynx. Echocardiography, which had been performed at the time the hemiparesis was first noted, suggested a thrombus in his left ventricle, presumably secondary to his myocardial infarct. It was believed that he had probably suffered from a cerebral embolus from this thrombus.

He was eventually transferred from the ICU to the rehabilitation unit, where he gradually improved. When discharged home after 77 days in hospital, mobility, mental alertness and swallowing had all returned to normal. A cardiac ultrasound performed at that time showed markedly reduced left ventricular function. When last seen, 8 months after his initial illness, he had minimal residual left-sided weakness, but his exercise tolerance remained poor. This was thought to be due to poor left ventricular function, rather than residual respiratory disease.

### Case 3

A 33-year-old woman, who was 12 weeks pregnant, was admitted to the infectious diseases unit with a 4-day history of a rash typical of chickenpox, and a one-day history of increasing shortness of breath, cough and pleuritic chest pain. A chest X ray again showed changes compatible with chickenpox pneumonia. All three of her children had recently had chickenpox and her husband had just recovered from varicella encephalitis. She was also a smoker.

She was immediately started on acyclovir 10 mg/kg three times daily and cefuroxime, and transferred to our ICU. Tracheal intubation and artificial ventilation were required because of rapid deterioration in respiratory function. In spite of this there was a problem in maintaining oxygenation. Over the next 24 hours her oxygen saturation decreased to below 80%, despite an inspired oxygen of 100%, and a PEEP of +7.3 mmHg (Fig. 2).

The prognosis was poor with this severity of hypoxaemia. However, as her respiratory failure was potentially reversible, it was decided to institute extracorporeal membrane oxygenation (ECMO). Veno-venous perfusion was used, as shown diagrammatically in Figure 3, using a Ski-Med membrane oxygenator and a Bio Medicus centrifugal pump. Heparin was infused to keep the activated clotting time at approximately 200 seconds. Flow through the oxygenator was initially set at 5 litres/minute.

On initiation of ECMO, rapid improvement in oxygen saturation occurred (Fig. 2). ECMO was continued for 90 hours, and the flow through the oxygenator and the inspired oxygen concentration were reduced slowly as lung function improved (Fig. 4). During this time 8 units of blood were required to keep her haematocrit at approximately 35%. At 90 hours, the flow through the oxygenator

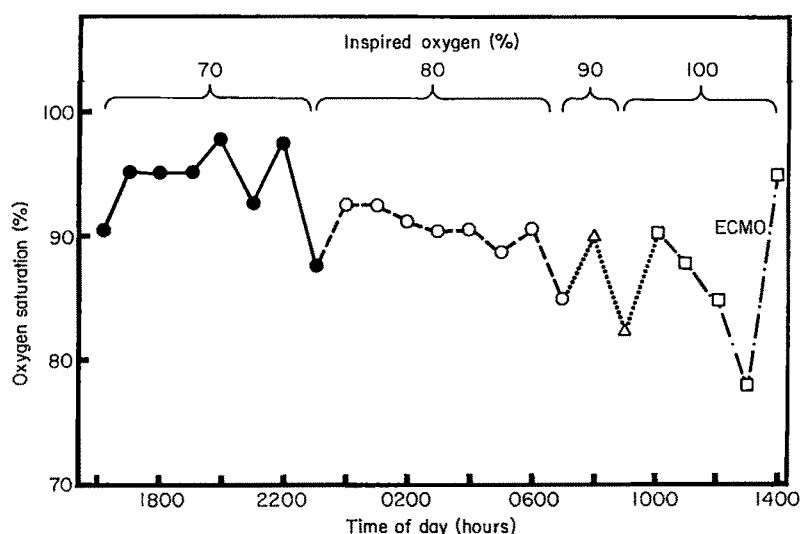


Fig. 2. Case 3, showing decreasing oxygen saturation, despite an increasing inspired oxygen concentration, over the first 24 hours.  $O_2 = 70\%$ , ●—●;  $O_2 = 80\%$ , ○—○;  $O_2 = 90\%$ , △—△;  $O_2 = 100\%$ , □—□.

was 2 litres and the  $P_{aO_2}$  was 16.7 kPa on an inspired oxygen of 50%. ECMO was then discontinued.

Following this, gradual improvement occurred, and artificial ventilation was discontinued on day 15 of her illness. Elective termination of pregnancy was carried out on day 21 because there was concern about the possibility of fetal malformation, due either to the varicella infection itself,<sup>3</sup> or to severe hypoxaemia. She was discharged home after 28 days in hospital. At 6 months follow-up, she was asymptomatic, had a normal chest X ray, and pulmonary function tests were normal, except for the transfer factor, which was decreased by 30%.

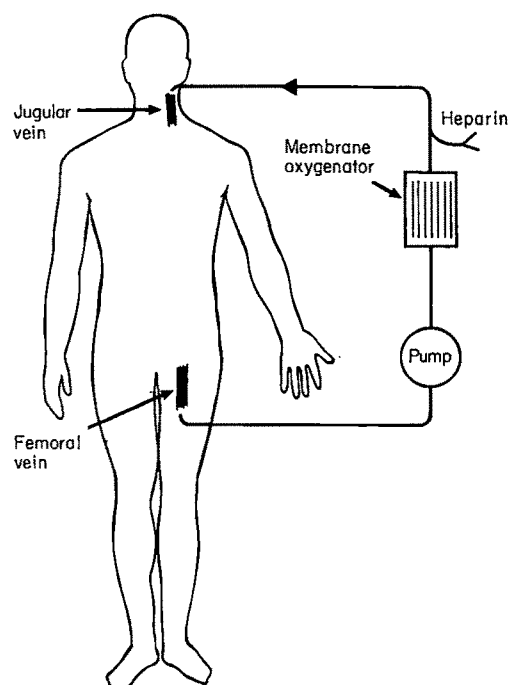


Fig. 3. A diagrammatic representation of the ECMO system used in Case 3.

### Discussion

Varicella or chickenpox is a common viral illness of childhood caused by the deoxyribonucleic acid (DNA) varicella-zoster virus. Although pneumonia does occur as a complication in previously healthy children,<sup>4</sup> it is far more common in the immunocompromised and in adults;<sup>5</sup> pregnant women are particularly at risk. Whilst this complication is associated with a significant morbidity and mortality, it does vary in severity from an asymptomatic radiological diagnosis to a life-threatening pneumonitis with respiratory failure.<sup>2</sup> The pneumonia characteristically develops within one week of the rash, with tachypnoea and cough, sometimes proceeding to hypoxia and respiratory failure in severe cases.<sup>6</sup>

Physical signs, other than those of hypoxia, are a poor guide to severity.<sup>2</sup> The risk of developing respiratory failure requiring artificial ventilation is uncertain, although in a small series described by Davidson *et al.*,<sup>7</sup> six out of 13 patients admitted to hospital were treated with IPPV. The exact mortality is also unknown, since chickenpox is not a notifiable disease, but figures of 15–20% have been quoted.<sup>8</sup> In pregnancy, however, the mortality from pneumonia is 30–41%.<sup>8,9</sup>

Smoking seems to be an important factor in the development of pneumonia. If the figures from two series are combined,<sup>10,11</sup> 23 out of 53 patients who smoked developed pneumonia, whereas only one out of 43 non-smokers did. All three of our patients were either moderate or heavy smokers.

The use of acyclovir in varicella pneumonia is recommended widely.<sup>8,12–18</sup> However, this advice is usually given case reports in which patients who have been treated with acyclovir have survived. As far as we are aware, there have been no clinical trials which show a definite benefit to patients who have already developed pneumonia. Davidson *et al.*,<sup>7</sup> in a series of 13 patients, suggested that antiviral therapy with acyclovir or vidarabine did not alter the clinical course of the disease once pneumonia had occurred. Balfour<sup>19</sup> in a report of varicella in eight immunocompromised children, suggested that acyclovir had no effect



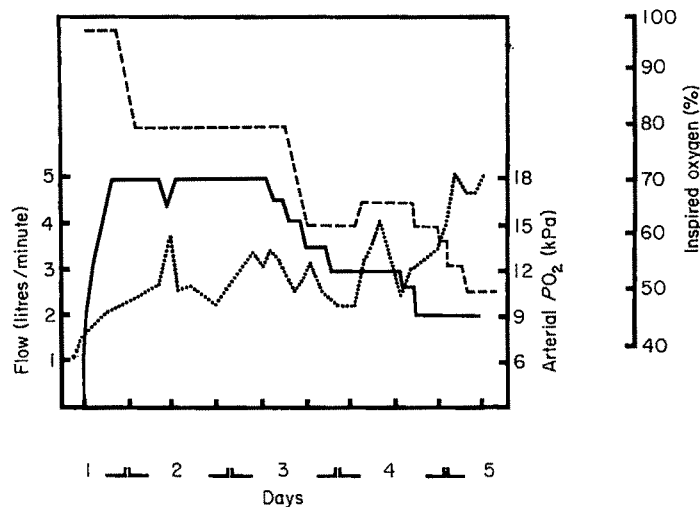


Fig. 4. Case 3, showing flow through the ECMO circuit —; inspired  $O_2$  concentration — — —; and arterial  $PaO_2$  ·····, over 90 hours showing improving oxygenation, allowing a decrease in inspired oxygen concentration and flow through the oxygenator.

once the disease had spread to the lungs or other organs. However, treatment with acyclovir is relatively safe. Renal problems can be avoided by ensuring adequate hydration, and monitoring the renal function.<sup>20</sup> There is no evidence that acyclovir is teratogenic,<sup>17</sup> and it might therefore be safe in pregnancy. It could possibly be recommended on the basis that it is unlikely to do any harm and it might do some good, except that it is an expensive drug. An alternative strategy might be to select patients at risk (i.e. adults, or even only adults who smoke, and immunocompromised children) and treat them with acyclovir at the first sign of chickenpox, before complications develop.

Myocarditis and arrhythmias, hepatitis and encephalitis are other well-recognised complications of chickenpox.<sup>21–23</sup> Our second patient suffered a variety of complications, although not all of them could be directly ascribed to the chickenpox. Although a vasculitis of cerebral vessels causing cerebral infarction has been described,<sup>24</sup> the most likely cause of his myocardial infarction was hypoxaemia, rather than a vasculitis of his coronary vessels. He was a heavy smoker, and although relatively young, he could have suffered from asymptomatic coronary artery disease. The arrhythmias were presumably secondary to his infarct, rather than the result of a myocarditis. The cause of the hemiplegia is, however, open to debate. An echocardiogram performed at the time suggested the possibility of a thrombus in the left ventricle. An embolus from this was thought to be the most likely cause of his hemiplegia. However, there have been two reports of cerebral infarction in patients with chickenpox<sup>24,25</sup> which have been attributed to vasculitis in the cerebral vessels. As suggested previously, the prolonged pyrexia without evidence of infection might have been a manifestation of varicella encephalitis. However, this diagnosis was purely speculative, and based on exclusion. The patient did show evidence of varicella hepatitis, with jaundice, raised plasma transaminases, and an ultrasound of his liver which was suggestive of inflammation.

We believe that in our third case, ECMO was life-saving. We were unable to find any reference to the use of ECMO in pregnancy in the English language literature. There has been a report of its successful use in a 5-year-old boy with

leukaemia and chickenpox pneumonia.<sup>26</sup> Unlike our patient, he still had moderately restrictive lung disease after 18 months. The mortality is very high in patients with severe respiratory failure, in whom conventional IPPV is failing to prevent life-threatening hypoxaemia, and ECMO is used as a last resort. Survival rates of 8–17% have been reported.<sup>26–28</sup> Our patient could be considered as an ideal candidate for ECMO: survival was unlikely without it; she had self-limiting lung disease whose pathology was potentially reversible; she had no evidence of sepsis; and no other organs had failed. We would recommend that ECMO should be considered in any patient with chickenpox pneumonia in whom conventional IPPV is failing to prevent severe hypoxaemia.

#### Acknowledgments

We are grateful to Dr McKendrick, Dr Hughes, Professor Monroe and Mr Goiti for permission to report their patients. We also thank the Department of Medical Illustration, Northern General Hospital for preparation of the figures.

#### References

1. WEBER DM, PELLECCIA JA. Varicella pneumonia. Study of prevalence in adult men. *Journal of the American Medical Association* 1965; **192**: 572–3.
2. HOCKBERGER RS, ROTHSTEIN RJ. Varicella pneumonia in adults: a spectrum of disease. *Annals of Emergency Medicine* 1986; **15**: 931–4.
3. PREBLUD SR, COCHI SL, ORENSTEIN WA. Varicella-zoster infection in pregnancy. *New England Journal of Medicine* 1986; **315**: 1416–7.
4. BULLOWA JGM, WISHIK SM. Complications of varicella. Their occurrence in 2,534 patients. *American Journal of Diseases of Children* 1939; **49**: 923–32.
5. JOSEPH CA, NOAH ND. Epidemiology of chickenpox in England and Wales, 1967–85. *British Medical Journal* 1988; **296**: 673–6.
6. TRIEBWASSER JH, HARRIS RE, BRYANT RE, RHOADES ER. Varicella pneumonia in adults. Report of seven cases and a review of literature. *Medicine* 1967; **46**: 409–23.
7. DAVIDSON RN, LYNN W, SAVAGE P, WANSBOROUGH-JONES MH. Chickenpox pneumonia; experience with antiviral treatment. *Thorax* 1988; **43**: 627–30.

8. HAWKINS G, GILSTRAP L, PATTERSON A. Acyclovir treatment of varicella pneumonia in pregnancy. *Critical Care Medicine* 1987; **15**: 336-7.
9. HARRIS RE, RHOADES ER. Varicella pneumonia complicating pregnancy. Report of a case and review of literature. *Obstetrics and Gynecology* 1965; **25**: 734-40.
10. GRAYSON ML, NEWTON-JOHN H. Smoking and varicella pneumonia. *Journal of Infection* 1988; **16**: 312.
11. ELLIS ME, NEAL KR, WEBB AK. Is smoking a risk factor for pneumonia in adults with chickenpox? *British Medical Journal* 1987; **294**: 1002.
12. LHEUREUX PH, DOUTRELEPONT JM, ASKENASI R. Varicella pneumonia in adulthood; acyclovir therapy may be of benefit. *Annals of Emergency Medicine* 1987; **16**: 1097.
13. EDER SE, APUZZIO JJ, WEISS G. Varicella pneumonia during pregnancy. Treatment of two cases with acyclovir. *American Journal of Neonatology* 1985; **5**: 16-8.
14. LANDSBERGER EJ, HAGER WD, GROSSMAN JH. Successful management of varicella pneumonia complicating pregnancy. A report of three cases. *Journal of Reproductive Medicine* 1986; **31**: 311-4.
15. VAN DE MEER JWM, THOMPSON J, TAN WD, VERSTEEG J. Treatment of chickenpox pneumonia with acyclovir. *Lancet* 1980; **2**: 473-4.
16. BOYD K, WALKER E. Use of acyclovir to treat chickenpox in pregnancy. *British Medical Journal* 1988; **296**: 393-4.
17. BROWN ZA, BAKER DA. Acyclovir therapy during pregnancy. *Obstetrics and Gynecology* 1989; **73**: 526-31.
18. GLASER JB, LOFTUS J, FERRAGAMO V, MOOTABAR H, CASTELLANO M. Varicella-zoster infection in pregnancy. *New England Journal of Medicine* 1986; **315**: 1416.
19. BALFOUR HH. Intravenous acyclovir therapy for varicella in immunocompromised children. *Journal of Pediatrics* 1984; **104**: 34-6.
20. BALFOUR HH. Acyclovir therapy for herpes zoster: advantages and adverse effects. *Journal of the American Medical Association* 1986; **255**: 387-8.
21. FIDDLER GI, CAMPBELL RWF, POTTAGE A, GODMAN MJ. Varicella myocarditis presenting with unusual ventricular arrhythmias. *British Heart Journal* 1977; **39**: 1150-3.
22. EY JL, SMITH SM, FULGINITI VA. Varicella hepatitis without neurologic symptoms or findings. *Pediatrics* 1981; **67**: 285-7.
23. JOHNSON R, MILBOURN PE. Central nervous system manifestations of chickenpox. *Canadian Medical Association Journal* 1970; **102**: 831-4.
24. GIBBS MA, FISHER M. Cerebral infarction in an adult with disseminated varicella. *Bulletin of Clinical Neurosciences* 1986; **51**: 65-7.
25. KAMHOLZ J, TREMBLAY G. Chickenpox with delayed contralateral hemiparesis caused by cerebral angiitis. *Annals of Neurology* 1985; **18**: 358-60.
26. HICKS RE, KINNEY TR, RAPHAELY RC, DONALDSON MH, EDMUNDS LH, NAIMAN JL. Successful treatment of varicella pneumonia with prolonged extracorporeal membrane oxygenation in a child with leukemia. *Journal of Thoracic and Cardiovascular Surgery* 1977; **73**: 297-302.
27. EGAN TM, DUFFIN J, GLYNN MFX, TODD TRJ, DEMAJO W, MURPHY E, FOX L, COOPER JD. Ten year experience with extracorporeal membrane oxygenation for severe respiratory failure. *Chest* 1988; **94**: 681-7.
28. ZAPOL WM, SNIDER MT, HILL JD, FALLAT RJ, BARTLETT RH, EDMUNDS LH, MORRIS AH, PIERCE EC, THOMAS AN, PROCTOR HJ, DRINKER PA, PRATT PC, BAGNIEWSKI A, MILLER RG. ECMO in acute respiratory failure, a randomised prospective study. *Journal of the American Medical Association* 1979; **242**: 2193-6.

CASE REPORT

## Paediatric blood pressure and anaesthesia

C. M. P. MATHER

### Summary

*One percent of children have appreciably and consistently raised arterial blood pressure. A 7-year-old girl admitted for routine tonsillectomy, had unrecognised hypertension which put her at increased risk. Should anaesthetic practice take more note of paediatric blood pressures?*

### Key words

*Complications.*

*Hypertension; paediatric.*

*Anaesthesia; paediatric.*

The British Hypertension Society recently stated that it does 'not recommend' widespread blood pressure screening in children, only in particular selected groups.<sup>1,2</sup> The total incidence of hypertension in childhood is greater than 1%; severe hypertension occurs in 10% of these, with a high risk of morbidity and mortality if undetected and untreated.<sup>3</sup>

### Case history

A 7-year-old, 17 kg girl was admitted for routine tonsillectomy. She had no relevant past medical, surgical or family history, and the physical examination was completely normal. Her pre-operative blood pressure was recorded as 170/130 mmHg by the ward nurses, but was interpreted as being the result of her 'running around'.

EMLA cream was applied to both hands before operation. Anaesthesia was induced with halothane, the trachea intubated and the patient was allowed to breathe spontaneously nitrous oxide/oxygen and halothane. Her pre-intubation blood pressure of 140/90 mmHg (by Dinamap) and heart rate of 100 beats/minute (bpm) increased to 190/140 mmHg and 120 bpm respectively. First the inspired halothane was increased from 1.5% to 2.0% and 3 mg of papaveretum was given intravenously. As frequent ventricular ectopics occurred, halothane was replaced by isoflurane and her breathing gently assisted by hand. She was considered to have been too lightly anaesthetised. Despite apparently adequate anaesthesia and analgesia, her blood pressure remained 160/100 mmHg (both arms). The

surgeon commented on difficulty with haemostasis.

In the recovery room, she was conscious and pain free, but her blood pressure increased further to 170/130 mmHg. On examination her abdomen and femoral pulses were normal. Three hours later she was returned to the operating theatre because of renewed bleeding. This was controlled after an estimated blood loss of 500 ml, for which she received a blood transfusion. Her clotting screen had been normal.

In view of the previous events her lungs were ventilated electively and she was transferred to the ITU. Her hypertension (peak pressures recorded upto 240/150 mmHg by radial arterial line) continued despite papaveretum (0.14 mg/kg/hour) and midazolam (0.15 mg/kg/hour), and was therefore controlled with a labetalol infusion (0.9 mg/kg/hour). She was clinically well sedated.

After 36 hours the oropharyngeal pack was removed and her trachea was quickly extubated. Intravenous therapy was reduced as oral medications were commenced; on discharge she was receiving labetalol 100 mg three times a day, captopril 6.25 mg three times a day and chlorothiazide 200 mg daily.

On investigation, the abnormal findings were moderate left ventricular hypertrophy on ultrasound and arteriolar thickening on fundoscopy. Plasma renin was 36 ng/ml (normal is less than 2), aldosterone was 4899 pmol/litre (normal is less than 750) and an aortogram demonstrated an occluded left renal artery, with enlarged lumbar collaterals. Future treatment will involve either bypass grafts or nephrectomy.

C.M.P. Mather, MB, ChB, Registrar, Sir Humphry Davy Department of Anaesthesia, Bristol Royal Infirmary, Bristol BS2 8HW.

Accepted 15 May 1990.

### Discussion

The British Hypertension Society suggest that widespread screening in children is probably not justified because of the low frequency and great variability<sup>4,5</sup> of hypertension, and the unclear cost benefit ratio of treatment.<sup>1,2</sup>

The incidence of childhood hypertension is dependent on the definition used; an epidemiologically defined upper limit of normotension for adults such as 140/90 mmHg is not applicable and if defined by the 95th centile alone, 5% of all children will be designated hypertensive. The incidence of hypertension in children is reported between 1% and 3%.<sup>3,6</sup> Children also display the phenomenon of 'tracking' whereby an individual's blood pressure tends to remain in the same percentile of the distribution over a period of many years.<sup>7</sup>

Hypertension in children is defined and classified<sup>3</sup> into: *borderline*: greater than the 90th centile and occasionally greater than the 95th centile; *mild/moderate*: repeatedly greater than the 95th centile without end organ involvement; *severe*: repeatedly greater than the 95th centile plus 10–15 mmHg, or any pressure with end organ involvement. Fewer than one in five children with an initial recording above the 95th centile will have three repeated pressure measurements which consistently exceed the 95th centile.

Although the incidence of severe hypertension is less in children (0.1–0.5%) than in adults, the aetiology in 63–94% is secondary in origin,<sup>8</sup> whereas in adults the majority of causes are unknown (90% are primary/essential).

The major cause of hypertension in children is renal disease; secondary cardiovascular or endocrine pathology are less common.<sup>8</sup> Secondary causes can also be classified into acute, transient and usually short-lived causes (e.g. acute glomerulonephritis) and chronic (e.g. coarctations). The younger the patient and the greater the severity of the hypertension, the increased likelihood of a secondary, rather than primary aetiology.<sup>8</sup> A proportion of those above the 95th centile are simply obese and/or lacking exercise.<sup>8</sup>

The British Hypertension Society, although not in favour of widespread screening, advocate the following groups of children should have their blood pressure recorded: the sick; previously hypertensive; diabetic; those with neurofibromatosis and 'probably' those with a family history.

All children coming into hospital should have blood pressures recorded routinely for the following reasons:<sup>9,10</sup> there is a significant incidence of 1 in 100; it is 'cheap', convenient and easy to measure; hypertension is treatable/curable; detection avoids increased morbidity/mortality.<sup>3</sup>

In some children the diagnosis will not be in doubt, but if the blood pressure recordings are equivocal or labile, then repeat estimations may be made. The management of an equivocal case detected before routine surgery is more

difficult, because there is controversy amongst paediatricians themselves as to when and when not to treat.<sup>1</sup> Children with severe primary or secondary hypertension, or with symptoms or signs of end organ damage are likely to benefit from investigation and treatment.

What is the present anaesthetic practice? Few anaesthetists, look for a child's blood pressure before elective surgery, and yet they would always check a young adult's. For those anaesthetists who do check a child's blood pressure and find an abnormal result, it is easy to dismiss it as the consequence of an active or anxious child, or the use of an incorrect cuff. There will also be failures caused by poor cooperation, but hypertension will probably be recognised increasingly in the young.<sup>11</sup>

This particular child was put at increased risk because of the common practices of either not checking, or of ignoring, or of the 'explaining away' of paediatric blood pressures.

### Acknowledgment

I am grateful to Mr P.G. Bicknell, FRCS, for allowing me to report his case, and to Professor Prys-Roberts for his advice.

### References

1. DE SWIET M, DILLON MJ, LITTLER W, O'BRIEN E, PADFIELD PL, PETRIE JC. Measurement of blood pressure in children (Recommendations of a working party of the British Hypertension Society). *British Medical Journal* 1989; **299**: 497.
2. DE SWIET M, DILLON MJ. Hypertension in children. *British Medical Journal* 1989; **299**: 469–70.
3. DILLON MJ. Modern management of hypertension. In: MEADOW R, ed. *Recent advances in paediatrics*, Vol. 7. Edinburgh: Churchill Livingstone, 1984: 35–53.
4. VOORS AW, WEBBER LS, FRERICHS RR, BERENSON GS. Body height and body mass as determinants of basal blood pressure in children. *American Journal of Epidemiology* 1977; **106**: 101–8.
5. DE SWIET M, FAYERS P, SHINEBOURNE EA. Blood pressure in 4 and 5-year-old children: the effects of environment and other factors in its measurement the Brompton study. *Journal of Hypertension* 1984; **2**: 501–5.
6. DILLON MJ. Investigation and management of hypertension in children. *Pediatric Nephrology* 1978; **1**: 59–68.
7. MILLER D, FARMER R. Hypertension. Incidence, prevalence and mortality. In: *Epidemiology of diseases*. Oxford: Blackwell Scientific, 1982: 124–6.
8. LONDE S. Causes of hypertension in the young. *Pediatric Clinics of North America* 1978; **25**: 55–65.
9. DE SWIET M, FAYERS P, SHINEBOURNE EA. The value of repeated blood pressure measurements in children—the Brompton study. *British Medical Journal* 1980; **280**: 1567–9.
10. HART JT. Hypertension in childhood and youth. In: *Hypertension*, 2nd edn. Edinburgh: Churchill Livingstone, 1987: 202–13.
11. KAPLAN N. Hypertension in childhood and adolescence. In: *Clinical hypertension*, 3rd edn. Baltimore: Williams and Wilkins, 1982: 411–33.

## Computer-controlled anaesthesia in the management of bronchopleural fistula

J. A. DONNELLY AND R. E. WEBSTER

### Summary

*The anaesthetic management of a bronchopleural fistula using a computer-controlled propofol infusion system is described.*

### Key words

*Anaesthesia; intravenous, propofol.*

*Equipment; computers, infusion pumps.*

*Complications; bronchopleural fistula.*

The choice of induction of anaesthesia in the presence of a bronchopleural fistula presents a problem. Bronchial intubation is essential to isolate the diseased lung from the healthy lung, to prevent contamination of the latter with infected material from the pleural space and to prevent failure of ventilation from the large air leak through the chest drain. The conventional choice is between an inhalational and a rapid sequence induction. Both techniques have problems. An inhalational induction with the patient in a semi-upright position is a difficult technique; it may cause cardiovascular problems or induce coughing and therefore contamination of the other lung. A rapid sequence induction risks the failure of bronchial intubation if intubation proves difficult. We present a case in which an alternative method for the induction of anaesthesia in this condition was used.

### Case history

The patient, a 62-year-old man, had undergone a right upper lobectomy 5 months before for carcinoma of the lung. Past medical history included squamous cell carcinoma of the right tonsil 11 years before, treated by radical neck dissection, right hemimandibulectomy and radiotherapy. The mandible was reconstructed 5 years later with an iliac crest graft.

The patient developed a persistent air leak after his lobectomy which eventually resolved. He was discharged from hospital one month after operation. On discharge he was known to have incomplete expansion of the right lung and a persistent cough. His condition remained stable for 5

months, at which time his cough increased and he developed increasing dyspnoea and right axillary chest pain. A chest X ray demonstrated no further expansion of his right lung and the presence of fluid in the right pleural cavity which was thought to be infected. A right thoracotomy was performed for removal of the pyogenic membrane. The lung did not expand fully after operation and 4 days later he developed surgical emphysema of the right chest wall; a chest film confirmed an enlarged pneumothorax. A further drain was inserted but despite normal medical treatment the lung failed to expand and an air leak persisted. The decision was made to close this surgically.

The patient was pre-oxygenated for 5 minutes with 100% oxygen and anaesthesia was induced using a computer-controlled propofol infusion,<sup>1</sup> which started at a predicted blood level of 5 µg/ml. Spontaneous respiration was maintained and a careful laryngoscopy performed. Anaesthetic depth was judged to be insufficient for intubation and therefore the predicted blood level of propofol was increased to 6 µg/ml. Again spontaneous respiration was maintained and the patient's trachea intubated with a 39-G left-sided Mallinckrodt double-lumen tube. Spontaneous respiration continued with little interruption after intubation and there were no episodes of apnoea. No coughing was seen and therefore no contamination of the healthy lung was thought to have occurred. Once the position of the tube was checked, muscle relaxation was achieved with 5 mg vecuronium, and intermittent positive pressure ventilation was started. Monitoring during induction included pulse oximetry, automatic noninvasive blood pressure and

J.A. Donnelly, BSc, MB, ChB, Registrar, R.E. Webster, MB, ChB, FFARCS, Consultant, Department of Anaesthesia, Ninewells Hospital and Medical School, Dundee DD1 9SY.

Accepted 30 August 1990.

an ECG. Invasive monitoring was established immediately after induction, with a radial arterial line and an internal jugular central venous line.

Oxygen saturation did not decrease below 97% at any stage and the only significant cardiovascular change was moderate hypotension immediately after induction with a decrease to 80 mmHg systolic from a pre-operative value of 110 mmHg. This was assumed to be because of a combination of relative dehydration and the effects of propofol and was rapidly corrected with an infusion of colloid and a decrease in the predicted blood level of propofol to 4 µg/ml. Anaesthesia was maintained with a 50% nitrous oxide/oxygen mixture, vecuronium increments as required and the propofol infusion to keep the predicted blood level at 4 to 5 µg/ml. Total operating time was 3.5 hours and was uneventful except for one episode of atrial arrhythmia during surgical manipulation of the mediastinum.

The total volume of propofol infused throughout was 211 ml. The patient was transferred to the intensive care unit for postoperative care. Good analgesia was achieved with an interpleural catheter,<sup>2</sup> which was inserted intra-operatively and 0.5% bupivacaine boluses were given as required.

#### Discussion

A computerised delivery system which incorporates a mathematical model of the pharmacokinetics of propofol was used for this case.<sup>1</sup> It is based on a microcomputer (the

POS 200 version of the Psion Organiser) which controls an Ohmeda 9000 syringe driver. This portable system allows the anaesthetist to infuse propofol to the required blood concentration and maintain this level or alter it rapidly and easily. Compared to an inhalational induction there is very little risk of cardiovascular compromise and it is a more pleasant experience for the patient. Spontaneous respiration is maintained, so there is less risk of failure of ventilation from a large air leak if intubation is unsuccessful than with a rapid sequence induction using a muscle relaxant. Depth of anaesthesia is easily controlled and therefore intubation can be performed without coughing and resultant contamination of the healthy lung or apnoeic episodes which would require assisted ventilation. Maintenance of anaesthesia is achieved with continuation of the propofol infusion and recovery is rapid once the infusion is discontinued. The good quality of recovery allows early extubation; this reduces the risks of high airway pressures which may occur if the patient requires postoperative ventilation.

We consider this technique is a useful alternative to the conventional management of induction of anaesthesia in the presence of a bronchopleural fistula.

#### References

1. KENNY GNC, WHITE M. A portable computerised infusion system for propofol. *Anaesthesia* 1990; **45**: 692-3.
2. COVINO BG. Interpleural regional analgesia. *Anesthesia and Analgesia* 1988; **67**: 427-9.



CASE REPORT

## Traumatic pneumocephalus following head injury

### A complication of general anaesthesia

M. D. FINCH AND G. A. R. MORGAN

#### Summary

*A 17-year-old patient who developed a tension pneumocephalus following a general anaesthetic 29 days after a severe head injury is described.*

#### Key words

*Complications; accident, pneumocephalus*

#### Case history

A 17-year-old male motor-cyclist was admitted to hospital following a road traffic accident. In the Accident and Emergency Department he was awake but irritable with a Glasgow Coma Score of 11. He had bilateral peri-orbital oedema. His pupils were unequal, but exhibited intact light reflexes. The fundi were normal. There was no obvious CSF rhinorrhoea or otorrhoea, although fresh blood was present in both nares. His other injuries were a fracture of the right wrist and an area of skin loss on the right thigh. Skull radiography revealed a right frontal fracture extending into the ethmoid sinus (Fig. 1).

A general anaesthetic was administered to allow safe transfer to the CT scanner. The technique involved pre-oxygenation during 3 minutes of spontaneous ventilation, the administration of fentanyl 100 µg intravenously, followed by a rapid sequence induction with cricoid pressure using thiopentone 250 mg and suxamethonium 75 mg. His trachea was intubated with a 9.0-mm cuffed oral tracheal tube. Atracurium 50 mg and midazolam 8 mg were given intravenously to allow ventilation during the transfer. ECG, pulse oximetry and noninvasive blood pressure were monitored continuously. CT scan revealed generalised cerebral oedema with ventricular compression. There was an area of contusion in the right frontal region and a similar contusion of the left occipital lobe, suggestive of a contre-coup injury. There was a fracture of the frontal bone extending into the right orbit. Blood was present in the right maxillary antrum. There was no evidence of intracranial air.

The patient was transferred to the Intensive Care Unit

where ventilation was continued for 48 hours, after which his trachea was extubated. His initial satisfactory level of consciousness gradually deteriorated over the following 8 hours, necessitating further ventilation. A second CT brain scan showed persistence of the cerebral oedema. Again, there was no evidence of intracranial air. Artificial ventilation was continued on the Intensive Care Unit for a further 48 hours. Extubation was then followed by satisfactory awakening and recovery.

The following day the patient received an interscalene brachial plexus block to allow manipulation of his wrist fracture. Six days later he was well enough to transfer to the Rehabilitation Unit. Continued progress over the following 15 days restored him to an independent existence in most respects.

Eighteen days after discharge from Intensive Care, anaesthesia was arranged for a split skin graft to the wound on his right thigh. The planned spinal technique was abandoned because the anaesthetist was unable to locate the subarachnoid space. General anaesthesia was administered using intravenous fentanyl 100 µg followed by propofol 150 mg. A size 4 laryngeal mask airway was inserted and the patient allowed to breathe spontaneously a mixture of oxygen (33%), nitrous oxide (66%) and enflurane. ECG, pulse oximetry and noninvasive blood pressure were monitored throughout. The anaesthetic lasted 30 minutes followed by satisfactory awakening in the recovery room.

The patient returned to the Rehabilitation Unit where it was noted the following day that he was still sleepy. Over the next 3 days his level of consciousness gradually deteriorated. He complained of frontal headaches and nausea, and subsequently became disorientated and confused,

---

M. D. Finch, MB, ChB, Senior House Officer, Intensive Care, G. A. R. Morgan, FFARCS, Consultant, Intensive Care Unit, Royal Cornwall Hospital (Treliske), Truro, Cornwall TR1 3LJ.

Correspondence should be addressed to Dr G. A. R. Morgan please.

Accepted 20 July 1990.



Fig. 1. X ray skull showing right frontal fracture extending into the ethmoidal sinus.

progressing to loss of vocalisation and continence. Mild papilloedema of the left optic fundus was observed, suggesting raised intracranial pressure. A third CT brain scan revealed a large intraventricular and multiple smaller subarachnoid aerocoeles. Air was present over the surface of the brain, with a few bubbles adjacent to the medulla. The lateral ventricles were dilated and exhibited fluid levels (Fig. 2).

The patient was transferred to the subregional neurosurgical unit where a large dural tear in the roof of the right ethmoid was repaired. He subsequently returned to our Rehabilitation Unit where he continued to make an excellent recovery and was discharged home.

#### Discussion

Traumatic pneumocephalus or intracranial aerocoele is a complication of skull fractures which result in a communi-

cation between an air-containing cavity and the interior of the cranium. It may result from fractures involving the frontal, ethmoid or sphenoid sinuses, or the mastoid air cells. There is usually cerebrospinal fluid (CSF) rhinorrhoea or otorrhoea because of the associated dural tear.

Patients are at risk of developing a tension pneumocephalus if air is forced through the dural tear into the cranium under pressure. For this reason, patients with a skull fracture and CSF rhinorrhoea should be instructed not to blow their noses.

Air within the cranium may lead to neurological dysfunction including altered conscious level, confusion, convulsions, aphasia and hemiparesis. Symptoms may vary in severity and may be relieved by an episode of CSF rhinorrhoea. Alternatively, the presence of air may be asymptomatic until signs of raised intracranial pressure supervene. A tympanic note may be heard on skull percussion and a succussion splash may be heard by both patient

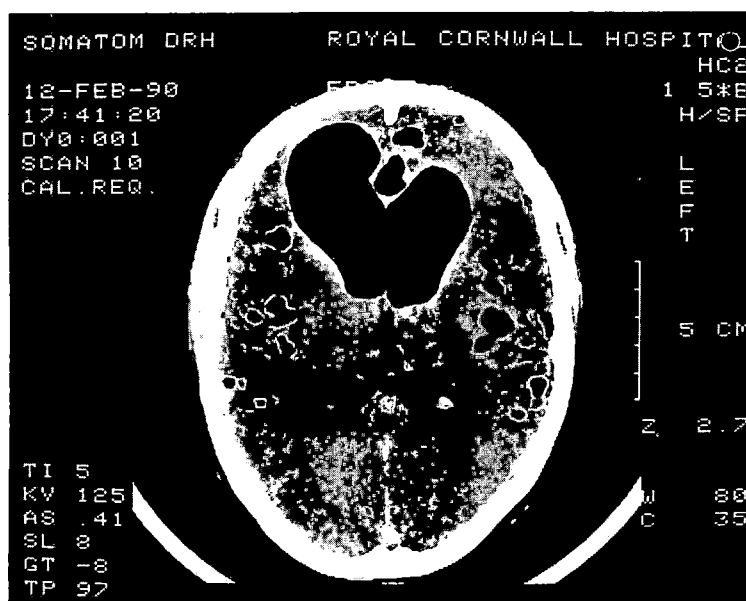


Fig. 2. CT brain scan showing a large intraventricular and smaller subarachnoid aerocoeles. The lateral ventricles are dilated and there is air over the brain surface.

and examiner when the patient shakes his/her head.<sup>1</sup> The definitive diagnosis is made by skull radiography or CT scan.

The effects of nitrous oxide anaesthesia on intracranial air are well documented. During pneumo-encephalography nitrous oxide diffuses into the air-filled ventricles because of its greater solubility in blood than nitrogen.<sup>2</sup> Nitrous oxide diffusion may double the size of an air-filled cavity within several minutes,<sup>3</sup> but this does not preclude its use during general anaesthesia for pneumoencephalography when intracranial pressure is normal, because the increase in intracranial pressure is balanced by drainage of cerebrospinal fluid. Nitrous oxide anaesthesia may produce dangerous increases in intracranial pressure<sup>2,4</sup> when it is elevated before pneumoencephalography, but the effect of nitrous oxide diffusion can be reduced during pneumoencephalography by the use of intrathecal nitrous oxide as the contrast gas.<sup>4</sup> However, following posterior fossa surgery, significant tension pneumocephalus was reported because of nitrous oxide diffusion after dural closure when intracranial pressure was previously low.<sup>5,6-8</sup> It is reasonable to expect that similar effects could occur when intracranial air is introduced by other means, for example, trauma.

In our case it is possible that some air was present before operation as a result of sneezing or nose-blowing. Postoperative deterioration could then be attributed to nitrous oxide diffusion. However, air in the subarachnoid space may be expected to be reabsorbed in 7–14 days. It is also possible that air was introduced during the attempted spinal anaesthetic.<sup>9,10</sup>

We believe that the likely cause was insufflation of oxygen or an oxygen, nitrous oxide mixture into the patient's oropharynx before insertion and during inflation of the cuff of the laryngeal mask airway, before an airtight seal was obtained. This could have forced gases into the cranial cavity via the ethmoid dural tear. The combination of fentanyl and propofol is highly likely to have produced a period of apnoea during which insufflation was necessary. The increase in intracranial pressure would have been exacerbated by hypercarbia and cerebral vasodilation in this spontaneously breathing patient.

In patients susceptible to formation of a tension pneumocephalus we would suggest the following. A skull X ray should be part of the pre-anaesthetic assessment to exclude intracranial air.<sup>8</sup> The anaesthetic technique should avoid

application of gases under pressure to the oro- or nasopharynx. If pressurisation of the oro- or nasopharynx is unavoidable, the presence of intracranial air should be excluded by skull X ray and a nitrous oxide-free anaesthetic should be administered. Failure of recovery from anaesthesia, vomiting in the postoperative period or later deterioration in conscious level is an indication for skull radiography to exclude pneumocephalus.

It is difficult to know for how long after the injury these recommendations should be followed.<sup>11</sup> The ingress of air may be delayed for days or weeks. A patient with a communication between oropharynx and cranial cavity will continue to be at risk of pneumocephalus formation until the dura has healed or been repaired. Cessation of rhinorrhoea is no evidence of natural repair. Effective healing in patients with traumatic pneumocephalus should be referred for formal neurosurgical assessment.<sup>11</sup>

### References

1. WALTON J. *Brain's diseases of the nervous system*, 9th edn. Oxford: Oxford University Press, 1985.
2. SAIDMAN LJ, EGER EI II. Change in cerebrospinal fluid pressure during pneumoencephalography under nitrous oxide anesthesia. *Anesthesiology* 1965; **26**: 67–72.
3. MARTIN, DC. Nitrous oxide and pneumocephalus. *Neurosurgery* 1987; **21**: 270.
4. GORDON E, GREITZ T. Effect of nitrous oxide on cerebrospinal fluid pressure during encephalography. *British Journal of Anaesthesia* 1970; **42**: 2–8.
5. ARTRU AA. Nitrous oxide plays a direct role in the development of tension pneumocephalus intraoperatively. *Anesthesiology* 1982; **57**: 59–61.
6. KITAHATA LM, KATZ JD. Tension pneumocephalus after posterior-fossa craniotomy, a complication of the sitting position. *Anesthesiology* 1976; **44**: 448–50.
7. HULETT WB, LAING JW. Tension pneumocephalus and the sitting position. *Anesthesiology* 1976; **45**: 578.
8. ARTRU A, SOHN YJ, EGER EI II. Increased intracranial pressure from nitrous oxide five days after pneumoencephalography. *Anesthesiology* 1978; **49**: 136–7.
9. HEANEY GAH. An interesting dural tap. *Anaesthesia* 1984; **39**: 386.
10. RODERICK L, MOORE DC, ARTRU AA. Pneumocephalus with headache during spinal anesthesia. *Anesthesiology* 1985; **62**: 690–2.
11. LEWIN W. Cerebrospinal fluid rhinorrhea in nonmissile head injuries. *Clinical Neurosurgery* 1966; **12**: 237–52.

## Pulse oximetry compared with Doppler ultrasound for assessment of collateral blood flow to the hand

K. PILLOW AND I. A. HERRICK

### Summary

*Ischaemic injury to the hand after arterial cannulation is a rare but well documented complication and routine testing of the adequacy of collateral circulation is widely advocated. The widespread availability of the pulse oximeter in the operating theatre, its applicability in circumstances where the patient is unable to cooperate, and its dependence on pulsatile blood flow suggest that this device could potentially be usefully applied to the assessment of collateral blood flow. The reliability of the pulse oximeter to detect the presence or absence of collateral circulation was prospectively compared to Doppler ultrasound in 109 hands from 64 adult patients. Nine hands demonstrated inadequate ulnar collateral flow, one hand demonstrated inadequate radial collateral flow and a persistent median artery was found in one hand. In all patients the results of pulse oximeter testing (probe placed on the thumb) correlated precisely with the results obtained with the Doppler device (probe located over the lateral aspect of the superficial palmar arch). These results demonstrate pulse oximetry to be a reliable method of assessing collateral blood flow to the hand before arterial cannulation.*

### Key words

*Equipment; pulse oximeter, Doppler ultrasound.*

The use of radial arterial lines is now commonplace in the anaesthetic and postoperative care of critically ill patients. Ischaemic injury to the hand after radial artery cannulation is a rare but well documented complication<sup>1</sup> and many authors advocate routine testing of the adequacy of the collateral circulation to the hand before arterial cannulation.<sup>2–4</sup>

The modified Allen's test has achieved widespread popularity for assessing the adequacy of collateral circulation to the hand. However, this test requires an awake, cooperative patient and thus is not readily applicable to many critically ill patients and those under general anaesthesia. In contrast, the use of Doppler ultrasound does not require patient cooperation and several studies have demonstrated the reliability of this device for assessing collateral blood flow to the hand.<sup>4–6</sup> Doppler ultrasound, however, is not always readily available or convenient to use in the operating room.

Several clinical reports have suggested that the pulse oximeter is a useful and convenient adjunct to the Allen's test,<sup>7–11</sup> however, assessment of the reliability of this technique has been incomplete. Recently, Glavin *et al.*<sup>12</sup> reported finding no correlation between the results of the pulse oximeter or the pulse meter compared to Doppler

ultrasound for assessing collateral blood flow to the hand. Despite these latter findings, the dependence of the pulse oximeter on pulsatile blood flow should enable this device to detect collateral circulation. This study was designed to compare prospectively the reliability of the pulse oximeter to Doppler ultrasound for assessing collateral blood flow to the hand.

### Methods

Sixty-four adult patients scheduled for elective surgical procedures were studied after institutional ethics approval and acquisition of informed consent. Palmar circulation was assessed bilaterally in each patient, surgery permitting, and in all but three patients general anaesthesia was induced before testing. These three patients required pre-induction insertion of radial arterial lines so testing was conducted awake. Anaesthetic management, including the decision to place an arterial line, was conducted as appropriate for the surgical procedure involved. No standardised anaesthetic protocol was employed.

Testing of collateral circulation was conducted in accordance with a standardised protocol after induction of general anaesthesia. A pulse oximeter probe (Nellcor

K. Pillow, MD, FFARCSI, Senior Resident, I.A. Herrick, BSc, MD, FRCPC, Lecturer, Department of Anaesthesia, University Hospital, 339 Windermere Road, London, Ontario, Canada N6A 5A5.

Correspondence should be addressed to Dr I.A. Herrick please.

Accepted 3 September 1990.

**Table 1.** Assessment of collateral arterial flow: pulse oximetry compared with Doppler ultrasound.

Ulnar collateral flow			Radial collateral flow		
Pulse oximeter signal	Doppler signal		Pulse oximeter signal	Doppler signal	
	Present	Absent		Present	Absent
Present	100	0	Present	108	0
Absent	0	9	Absent	0	1

N-1000) was placed on the patient's thumb under stable haemodynamic conditions and the presence of a normal pulsatile waveform and haemoglobin saturation confirmed. A nondirectional Doppler ultrasonic flow detector (Parks Medical Electronics Inc.—model 811) equipped with a 10 MHz probe was applied to the wrist to confirm the presence or absence of radial and ulnar arterial flow. The transducer was then positioned in the web space between the thumb and index finger and moved slowly medially over the palm until the lateral aspect of the superficial palmar arch was identified and the Doppler signal maximised at this location. With the Doppler probe and pulse oximeter positioned, the radial and ulnar arteries were simultaneously occluded by digital compression at the wrist for approximately 15 seconds and the disappearance of both pulse oximeter and Doppler signals were noted. Occluding pressure over the ulnar artery was then released and each device monitored for the reappearance of the signal. The procedure was then repeated testing radial collateral flow during ulnar artery occlusion.

### Results

A total 109 hands were evaluated in the 64 patients studied. The mean age of the study population was 47 years (range 17–88 years). In all patients, the presence or absence of collateral blood flow detected by the pulse oximeter correlated precisely with the Doppler results (Table 1).

Nine hands were found to have inadequate ulnar collateral flow with loss of signal to both devices with the radial artery occluded. In two hands the inadequacy of ulnar collateral flow to the palm correlated with absence of an identifiable ulnar pulse at the wrist. In four other hands only a weak ulnar pulse could be identified at the wrist by palpation and/or Doppler testing. Three hands demonstrated a normal ulnar pulse at the wrist, but inadequate flow to the lateral superficial palmar arch and the thumb. These three patients presumably had incomplete superficial arches.

One patient demonstrated inadequate radial collateral flow with loss of signal to both the pulse oximeter and the Doppler following ulnar artery occlusion. A radial pulse was identified at the wrist of the involved hand.

One patient was found to have a persistent median artery in one hand. This patient failed to demonstrate a loss of signal with either device following simultaneous occlusion of both the radial and ulnar arteries. Subsequent Doppler testing identified a source of pulsatile median blood flow at the wrist.

### Discussion

The arterial blood supply to the hand is complex, and multiple anatomical patterns have been identified.<sup>4,13</sup> The

superficial palmar arch commonly provides the major source of blood flow to the hand (including the fingers) and typically arises as a continuation of the ulnar artery into the palm. The deep palmar arch is formed primarily by the radial artery and typically provides the major source of blood flow to the thumb and thenar eminence. Fortunately, the circulatory pattern in the hand is rarely as simple as the preceding description would imply and typically multiple anastomoses exist between the major collateral channels. This probably accounts for the very low incidence of ischaemic injuries observed following arterial cannulation.

Since the blood supply to the thumb tends to be radial artery dominant,<sup>3,13</sup> the thumb was selected as the site for the pulse oximeter probe to maximise the sensitivity of this test for inadequate ulnar collateral flow. Similarly, the Doppler probe was used to locate the superficial arch as close to the lateral margin of the hand as possible. Little *et al.*<sup>4</sup> who studied the circulatory patterns in normal hands with a Doppler flowmeter, found a higher incidence of inadequate ulnar collateral blood flow with the Doppler probe placed laterally compared to medially on the palm. Thus, placing the Doppler probe laterally on the palm should maximise the dependence of this device on radial artery flow and optimise its sensitivity to inadequate ulnar flow.

Under the conditions of this study an excellent correlation was found between the assessment of collateral circulation performed by the pulse oximeter compared with the Doppler ultrasound. These findings are clearly in contrast to those recently reported by Glavin *et al.*<sup>12</sup> in which no correlation was found between these two devices. The basis for this discrepancy in results is unclear. The pulse oximeter model differed between the studies and in our study all but three patients were tested under general anaesthesia. However, these factors are unlikely to account for the differences in results observed. Glavin *et al.*<sup>12</sup> placed the pulse oximeter probe on the index finger of their study patients, a site which may not be as sensitive to ulnar collateral blood flow as the thumb. Although they reported that radial and ulnar artery blood flow was confirmed by Doppler testing, the basis and location for establishing such flow was not specified. If the presence of blood flow was tested at the wrist, the findings would not necessarily correlate with blood flow results detected directly in the superficial arch. In our study, seven of the nine patients found to have inadequate ulnar collateral blood flow detected in the lateral superficial arch demonstrated normal (three) or weak (four) ulnar pulses at the wrist. Potentially, these differences in protocol may account for some of the discrepancy in results.

We found the pulse oximeter to be very sensitive to the presence of pulsatile blood flow and in many cases considerably more convenient to use than the Doppler in terms

of the manipulations required to maintain a satisfactory signal during testing. In our experience, the potential concern with the pulse oximeter is not related to its reliability at detecting collateral blood flow but rather the possibility that this device will detect collateral blood flow at levels which are inadequate to support tissues of the hand. This problem, however, is also associated with the use of a nondirectional Doppler.

Despite being widely advocated, testing of collateral circulation before arterial cannulation remains a controversial exercise since correlation between the results of currently available testing techniques and the risk of ischaemic injury has not been adequately confirmed.<sup>1</sup> While the results of this study demonstrate the pulse oximeter to be a reliable device for detecting collateral flow, the study is unable to correlate these results with the risk of ischaemic injury following cannulation of the radial artery. Nevertheless, based on reliability for detecting collateral blood flow, widespread availability in the operating room and convenience of use in unconscious patients, the results of this study suggest that the pulse oximeter represents a useful alternative to Doppler ultrasound and the modified Allen's test.

#### References

1. WILKINS RG. Radial artery cannulation and ischaemic damage: a review. *Anaesthesia* 1985; **40**: 896-9.
2. HUG CC. Monitoring: arterial cannulation. In: Miller RD, ed. *Anesthesia*, 2nd edn. Vol 1. New York: Churchill Livingstone, 1986; 435-7.
3. HUSUM B, PALM T. Arterial dominance in the hand. *British Journal of Anaesthesia* 1978; **50**: 913-6.
4. LITTLE JM, ZYLSTRA PL, WEST J, MAY J. Circulatory patterns in the normal hand. *British Journal of Surgery* 1973; **60**: 652-5.
5. MÖZERSKY DJ, BUCKLEY CJ, HAGOOD CO, CAPPS WF, DANNEMILLER FJ. Ultrasonic evaluation of the palmar circulation. *American Journal of Surgery* 1973; **126**: 810-2.
6. KAMIENSKI RW, BARNES RW. Critique of the Allen test for continuity of the palmar arch assessed by Doppler ultrasound. *Surgery, Gynecology and Obstetrics* 1976; **142**: 861-4.
7. NOWAK GS, MOORTHY SS, MCNIECE WL. Use of pulse oximetry for assessment of collateral arterial flow. *Anesthesiology* 1986; **64**: 527.
8. RAJU R. The pulse oximeter and the collateral circulation. *Anaesthesia* 1986; **41**: 783-4.
9. HOVAGIM AR, KATZ RI, POPPERS PJ. Pulse oximetry for evaluation of radial and ulnar arterial blood flow. *Anesthesia and Analgesia* 1988; **67**: S94.
10. LAUER KK, CHENG EY, STOMMEL KA, GUENTHER NR, KAY J. Pulse oximetry evaluation of the palmar circulation. *Anesthesia and Analgesia* 1988; **67**: S129.
11. DUNCAN PW. An alternative to Allen's test. *Anaesthesia* 1986; **41**: 88.
12. GLAVIN RJ, JONES HM. Assessing collateral circulation in the hand—four methods compared. *Anaesthesia* 1989; **44**: 594-5.
13. COLEMAN SS, ANSON BJ. Arterial patterns in the hand based upon a study of 650 specimens. *Surgery, Gynecology and Obstetrics* 1961; **113**: 409-24.



## Single operator cardiopulmonary resuscitation in ambulances

### Which ventilation device?

G. L. GREENSLADE

#### Summary

*Cardiopulmonary resuscitation en route to hospital is performed by a single-handed operator in many British ambulances. In this study, three emergency ventilation devices, and mouth-to-mouth breathing, were compared for effectiveness in unintubated patients. Seventeen paramedics used each method on a Laerdal manikin in a randomised order, under identical conditions. Three experienced cardiopulmonary resuscitation instructors repeated the tests in a moving ambulance. There were significant differences in minute volume ( $p < 0.01$ ) and number of effective chest compressions ( $p < 0.05$ ); mouth-to-mouth breathing produced the best overall results and the simplest device was a close second. The value of automatic ventilators for single-operator cardiopulmonary resuscitation in unintubated patients is questioned.*

#### Key words

*Ventilation; artificial.*

*Equipment; ventilators.*

*Heart; cardiac massage.*

Cardiopulmonary resuscitation (CPR) is performed by a single operator in many British ambulances. A variety of emergency ventilation devices is carried and automatic volume/time-cycled ventilators are frequently used for single-operator CPR in unintubated patients, a rare practice in hospitals.

Previous controlled studies have compared the performance of pocket masks with bag and mask sets, but only for artificial ventilation, as opposed to full CPR.<sup>1,2</sup> The author spent 5 years in emergency ambulance work, and encountered only three purely respiratory arrests, but over 300 cardiorespiratory arrests. This experience has been corroborated in discussions with other experienced paramedics. Therefore, studies that fail to evaluate these devices for full CPR are probably irrelevant to ambulance work.

This study compared the effectiveness of mouth-to-mouth, and three popular devices, when used by paramedic staff for single-operator CPR. Methods involving tracheal intubation were excluded because the majority of British ambulance crews cannot intubate.<sup>3</sup> Finally, a short trial was performed in a moving ambulance.

#### Methods

Seventeen Royal Navy paramedics performed CPR on a recording Resusci-Anne (Laerdal Medical Ltd) in a classroom. A new Wright's Respirometer (factory calibrated)

was attached to the dummy's exhaust port to measure expired gas volumes.

The following devices were tested: (1) Laerdal pocket mask with oxygen inlet (Laerdal Medical Ltd.); (2) Ambu Mk III resuscitator with transparent mask (Ambu International Ltd.); (3) PneuPAC automatic time/volume-cycled ventilator with adjustable tidal volume (PneuPAC Ltd.) using the transparent Ambu mask. This ventilator was set to deliver maximum tidal volume, with the air entrainment valve open, giving an inspired oxygen fraction ( $F_{iO_2}$ ) of about 0.45.

Each man started by performing CPR for 5 minutes using mouth-to-mouth ventilation, and aimed to follow the Resuscitation Council (UK)'s recommendations on technique.<sup>4,5</sup> Five minutes of rest were followed by a further 5 minutes of CPR, using one of the test devices. This alternating CPR/rest sequence continued until the subject had used all the devices. The order in which they were used was determined with random number tables. The following data were recorded for each ventilation method: total gas volume delivered; number of breaths delivered; total number of *effective* chest compressions. Mean minute volumes, mean tidal volumes and mean numbers of effective cardiac compressions were calculated from these data, which were examined using analysis of variance (ANOVA).<sup>6,7</sup> Logarithmic transform to the data was applied, when necessary, to comply with statistical assumptions used in ANOVA. An assessment of the frequency

G. L. Greenslade, MB, BS, DA, Senior House Officer, Department of Anaesthetics, Royal Naval Hospital, Haslar, Gosport, Hants PO12 2AA.

**Table 1.** Summary statistics for minute volume (litres) for 17 subjects.

Method	Mean	Standard deviation	Minimum	Maximum
Mouth-to-mouth	10.5	3.1	4.6	17.8
Mouth-to-mask	8.5	2.7	4.6	15.6
Ambu-bag	4.5	1.2	2.4	6.2
PneuPAC	4.7	1.0	3.2	6.4

with which minute volumes exceeded 5 and 7 litre thresholds was made using Cochran's method.<sup>8</sup> These were evaluated using Meddis<sup>9</sup> whenever this suggested significant differences.

The trials were repeated by the author and two senior instructors from the Hampshire Ambulance Service in a standard emergency ambulance whose speed did not exceed 40 miles per hour on semi-urban roads. This test was constrained by cost, so that it was only possible to form subjective impressions.

### Results

The results of the main study are summarised in Tables 1 and 2.

#### Minute volume

The exhaled air methods produced significantly higher minute volumes than the PneuPAC and Ambu ( $p < 0.01$ ). Using minute volume threshold values of 5 or 7 litres, the two oral methods reached or exceeded these values significantly more often than the Ambu or PneuPAC ( $p < 0.01$ ). No difference could be established between the two oral methods, nor between the PneuPAC and the Ambu.

#### Effective compressions per minute

The average values for mouth-to-mouth were significantly higher than for the PneuPAC or Ambu ( $p < 0.05$ ). The mouth-to-mask average value was significantly higher than the PneuPAC's ( $p < 0.05$ ). There were no significant differences between the oral methods, or between the PneuPAC and Ambu.

The performance of chest compressions requires the operator to be at the patient's side, rather than at the head end. Thus it proved impossible to use the conventional 'anaesthetist's' mask-holding grip. The technique illustrated was successful in overcoming this problem (Fig. 1).

None of the three experienced operators were able to perform adequate CPR in the moving ambulance test. Chest compressions were often dangerously violent, because the operator fell onto the patient's chest as the ambulance travelled over a bump. Inflations were delayed because the operator could not find the Ambu-bag, which was repeatedly thrown to the floor by the ambulance's motion while the operator was occupied with chest compressions. Vehicle noise made it difficult to hear the PneuPAC cycling, so that many inflations were lost to the atmosphere.

The pocket mask lacerated one operator's gingiva when the ambulance went over a pothole, forcing the rigid infla-

**Table 2.** Summary statistics for compressions per minute for 17 subjects.

Method	Mean	Standard deviation	Minimum	Maximum
Mouth-to-mouth	40.3	7.8	29	57
Mouth-to-mask	38.2	7.1	26	54
Ambu-bag	36.2	5.5	28	47
PneuPAC	33.1	5.4	25	44

tion port into his mouth. Mouth-to-mask ventilation using the Ambu mask was attempted and appeared effective and safer, because the soft rubber inlet ring cushioned the operator's mouth. A piece of adhesive surgical tape secured the mask to the dummy's cheek and stopped it from falling to the floor, but permitted it to be flipped back to give fast access to the mouth.

The ambulance was driven quite slowly, but all the operators found it difficult to perform CPR. They were alarmed to find themselves using the dummy's head and cervical spine as handholds to avoid falling. These problems of stability diminished at road speeds below 30 mph.

### Discussion

Effective CPR requires adequate ventilation of the lungs with adequate numbers of effective cardiac compressions. CPR using mouth-to-mouth ventilation is known to work, is easy to perform and needs no special equipment.

How can mouth-to-mouth ventilation be improved upon in an unintubated patient? Devices that increase the  $F_{IO_2}$  may improve the oxygenation of the blood.<sup>10</sup> Mouth-to-mouth adjuncts are popular because they reduce the frequency with which professional rescuers come into mouth-to-mouth contact with patients. Any device used to replace mouth-to-mouth breathing should be easy to use and not hamper the delivery of chest compressions. It should be possible to deliver minute volumes that will eliminate enough carbon dioxide to delay the onset of the severe acidosis that accompanies cardiac arrest.<sup>11</sup> If the

**Fig. 1.** Grip adopted to maintain mask seal and clear airway with operator positioned beside the patient.

minute volume in a resting man is about 7.5 litres,<sup>12</sup> an artificially-ventilated minute volume of 7 litres is probably a reasonable target during CPR, with 5 litres representing an acceptable minimum.

Mouth-to-mouth breathing in this study was equalled by the pocket mask, which has the advantage of allowing oxygen supplementation, whilst not relying totally on compressed gas supplies. Its transparent design permits visual monitoring of the mouth and the operator may use both hands to seal the mask and maintain airway patency. The elasticated headband helps to stop the mask from being lost during chest compressions, but allows rapid removal should the patient vomit or regurgitate.

Bag and mask sets are notoriously difficult for nonanaesthetists to use. Some workers have suggested that two individuals are required to operate this apparatus effectively, so that chest compression would require a third person.<sup>13</sup> The main problem with the Ambu-bag in the moving ambulance was the disruption caused to the CPR cycle by the bag falling to the floor and being lost. However, the Ambu can be used without a compressed gas supply and is mechanically reliable.

Theoretically, the PneuPAC should be easier to use than a bag and mask, since the operator has both hands available to manipulate the mask and airway. It has the advantage of delivering an oxygen-rich mixture. Unfortunately, the autonomous action of the machine required the operators to conform to its cycle, causing chest compressions to be lost. If the operator failed to achieve a good mask seal before the machine started its next inflation, part, or all, of the breath was lost to the atmosphere. The background noise in the moving ambulance worsened this problem by masking the PneuPAC cycling sounds. In addition, it cannot operate without a compressed gas supply.

Cardiac arrest is often associated with a full stomach so that, in unintubated patients, continuous airway surveillance is essential. Some ambulance services issue Clausen harnesses, but this may make the mask difficult to remove quickly if the patient vomits or regurgitates. If the ventilator were to attempt to deliver a breath at his point, the outcome could be fatal. The Ambulance Service's training manual warns of the increased aspiration risk when using automatic ventilators, yet describes their use with Clausen harnesses for single-operator CPR.<sup>14</sup>

Other workers have shown that portable automatic ventilators are probably the method of choice for the ventilation of intubated patients during transportation,<sup>15</sup> although these patients had spontaneous cardiac outputs. Thus, the problem appears not to be the ventilator itself, but the application for which it is being used. These ventilators will probably become increasingly valuable as more ambulance crews learn to intubate.

Comparison of the methods is complicated because the Ambubag and PneuPac avoid rebreathing, whereas the exhaled air methods do not. The impact of this may be reduced because the rescuer's deadspace gas enters the patient's alveoli first, while his end-tidal gas, with its higher CO<sub>2</sub> content, occupies the victim's deadspace. The use of supplemental oxygen with the pocket mask should further dilute the CO<sub>2</sub> concentration. However, when a lower minute volume is combined with a reduced number of effective chest compressions, it seems reasonable to infer that CPR is less likely to be effective. This was the case with the two mechanical devices.

This study was constrained by the need to use training dummies. Nevertheless the conditions were uniform for each method tested. It is possible that the Naval paramedics may not have been as experienced as some of their civilian counterparts. However the results obtained do seem to indicate that real differences exist in the effectiveness of these devices. It is worth noting that mouth-to-mask ventilation was a novel method for the subjects, because it had not been included in the basic training package at the time of this study, unlike the other two devices. This makes the mouth-to-mask results even more impressive, since they were achieved merely by allowing the subjects to read the instructions on the storage box. However, even the best performances failed to meet the ideal standard. The obvious answer would be to abandon the practice of single-operator CPR, but this will need time and investment in training. Meanwhile, patients will continue to be dependent on their single-handed, nonintubating attendants.

The moving ambulance tests did not achieve statistical significance, but the subjective impressions gained were supported by the results of the main study.

The relative costs are as follows: Laerdal pocket mask with oxygen inlet £15, Ambu-bag £90, pneuPAC £670 complete.

### Conclusions

Mouth-to-mouth respiration gave the best results for ventilation and effective cardiac compressions, but is unacceptable for regular use by professional rescuers. The Laerdal pocket mask with oxygen inlet was the most satisfactory of the devices tested, but may need modification to soften the mouthpiece for use in moving ambulances. The transparent Ambu mask, secured to the patient's cheek with adhesive tape, was an alternative to the Pocket mask, and provided a safer mouthpiece for the rescuer, but lacked the oxygen inlet.

This study confirmed that bag and mask sets are difficult for nonspecialists to use. Automatic ventilators, such as the pneuPAC, are probably best reserved for intubated patients.<sup>4,15</sup>

It is very difficult to provide adequate CPR in a moving ambulance, but the advanced training of ambulance crews allows them to provide definitive resuscitative therapy *before* transporting the patient to hospital; this is the treatment of choice.<sup>16,17</sup> Meanwhile, keeping the ambulance speed below 30 mph and using oxygen-supplemented mouth-to-mask ventilation, appear to offer the best chance of success in unintubated patients receiving single-operator CPR.

### Acknowledgments

I thank Dr R.J. Pethybridge, Principal Statistician at the Institute of Naval Medicine, Surg Lt Cdr S.Q.M. Tighe, FCAnaes, RN, Consultant Anaesthetist at the RNH Haslar for his advice and encouragement; Mr R. Howard MBE, Senior Technician, standardised the equipment. The ambulance tests and interviews were performed with Messrs. P. Smees and J. Luck, Instructors at the Hampshire Ambulance Training Centre, with the kind permission of Mr G. Adams, the Chief Ambulance Officer.

The views expressed are those of the author and do not

necessarily reflect the official policy of the MOD (Navy) or of the Hampshire Ambulance Service.

### References

1. HAMER M, HOWELLS TH, WATSON R. A survey of Resuscitation Ventilatory Aids. *Journal of the British Association for Immediate Care* 1986; **9**: 31-2.
2. Manually operated resuscitators. *Health Devices* 1974; **3**: 164-76.
3. *Norfolk Ambulance Service*. Extended Training Clinical Audit (UK Ambulance Services). Norfolk, 1989.
4. FISHER JM. Recognising a cardiac arrest and providing basic life support. In: EVANS TR, ed. *ABC of Resuscitation*. London: British Medical Journal, 1986: 1-4.
5. MARSDEN AK. Basic Life Support. Revised recommendations of the Resuscitation Council (UK). *British Medical Journal*; **299**: 442-445.
6. WINER BJ. *Statistical principles in experimental design*. New York: McGraw-Hill, 1962.
7. SCHEFFE H. *The analysis of variance*. New York: John Wiley, 1959.
8. COCHRAN WG. The comparison of percentages in matched samples. *Biometrika* 1950; **37**: 265-266.
9. MEDDIS M. Unified analysis of variance by ranks. *British Journal of Mathematical and Statistical Psychology* 1980; **33**: 84-98.
10. SAFAR P. Pocket mask for emergency artificial ventilation and oxygen inhalation. *Critical Care Medicine* 1974; **2**: 273-6.
11. CHAMBERLAIN DA. Ventricular fibrillation. In: EVANS, TR, ed. *ABC of resuscitation*. London British Medical Journal, 1986: 5-8.
12. WEST JB. *Ventilation/blood flow and gas exchange*. Oxford: Blackwell, 1977: 3.
13. ELLING R, POLITIS J. An evaluation of emergency medical technicians' ability to use manual ventilation devices. *Annals of Emergency Medicine* 1983; **12**: 765-8.
14. *National Health Service Training Authority*. Ambulance Service: Basic Training. Bristol: NHSTA, 1988.
15. BRAMAN SS, DUNN SM, AMICO CA, MILLMAN RP. Complications of intrahospital transport in critically ill patients. *Annals of Internal Medicine* 1987; **107**: 469-73.
16. WEAVER WD, COBB LA, HALLSTROM AP, FAHRENBRUCH C, COPASS MK, RAY R. Factors influencing survival after out-of-hospital cardiac arrest. *Journal of the American College of Cardiologists* 1986; **7**: 752-7.
17. EISENBURG M, BERGNER M, HALLSTROM AP. Paramedic programs and out-of-hospital cardiac arrest: II. Impact on community mortality. *American Journal of Public Health* 1979; **69**: 39-42.

## A new portable oxygen system using liquid oxygen

C. M. H. RAMAGE, S. S. KEE AND A. BRISTOW

### Summary

*Liquid oxygen provides both practical and financial advantages in comparison with compressed oxygen when transferring patients within and between hospitals. We describe a portable liquid oxygen system and evaluate its uses and shortcomings in this area.*

### Key words

*Equipment; liquid oxygen.*

*Oxygen therapy; portable.*

Compressed oxygen in cylinders has several disadvantages when used in the management of patients who need to be moved from one location to another, or are treated outside hospital. Cylinders are large and heavy. Partially full cylinders are often discarded in favour of a new cylinder to ensure that there is sufficient oxygen. Spare cylinders need to be obtained and stored, therefore space and personnel are required.

The use of liquid oxygen has several advantages. A given quantity of oxygen can be carried in a smaller and lighter container. A partially empty container can be refilled, avoiding waste and the need for spare cannisters. The cost of liquid oxygen is also considerably less than that of compressed oxygen in cylinders.

Until recently, liquid oxygen containers could only provide low flows of oxygen because of limitations imposed by the rate of vaporization of the liquid. This precluded their use for critically ill patients and for resuscitation. The Companion EMT system, however, can provide sufficient flows for these purposes, as well as pressurised oxygen to drive a variety of gas driven devices.

### General description

The system consists of a portable Companion and a stationary unit, which contains either 10, 20 or 30 litres of liquid oxygen and is filled from a reservoir. The stationary units can provide a supply of oxygen, but are more usually used to fill the portable systems.

The portable units (Fig. 1) consist of a stainless steel double-walled, vacuum-insulated container which holds 1.2

litres of liquid oxygen at 450 kPa. Liquid is vaporized and warmed by warming coils before being delivered by a flow control unit. The latter is an adjustable, fixed orifice type control valve, which is graduated from one to 15 litres per minute of gaseous oxygen. A fully open setting allows for gas driven equipment such as a ventilator to be used. There is a differential pressure contents gauge which measures the volume of liquid oxygen remaining.

The unit is filled by placing it in a recess on the top of the stationary unit. A separate fill valve is opened and liquid decants into the container, displacing gas.

### Assessment of the system

The portable containers have been used over a period of 6 months in the hospital's helicopter-based intensive care unit. The stationary units have also been used on fixed-wing aircraft for longer transfers when greater capacity is required. Several modifications have been made during this time.

The equipment's performance has been assessed at Saint Bartholomew's Hospital.

### Unit performance

The manufacturers provide a recommended performance envelope diagram (Fig. 2) which has been devised using the Pneupac portable ventilator. Using a Pneupac Transpac ventilator, the initial units, which operated at 350 kPa, gave the performance indicated in Table 1. Further modifications were undertaken by altering the relief valve settings

C.M.H. Ramage, BSc, MB, BS, FFARCS, Lecturer, S.S. Kee, MB, ChB, FFARCS, Lecturer, A. Bristow, MB, BS, FFARCS, Consultant, Department of Anaesthesia, St Bartholomew's Hospital, West Smithfield, London EC1.

Accepted 28 August 1990.

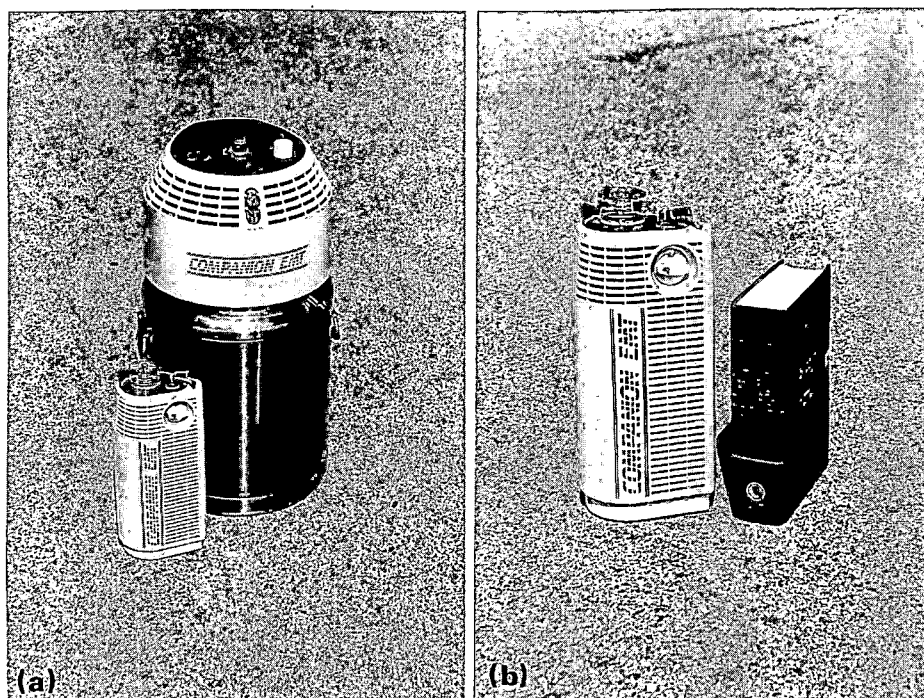


Fig. 1. (a) The portable and 20-litre liquid oxygen units. (b) A close up of the portable liquid oxygen unit.

thus allowing the units to operate at 450 kPa which provided the performance in column 3. These figures were obtained using an  $F_{IO_2}$  of 1.0. With the airmix or air entrainment, the indicated settings were achieved up to the maximum of 20 litres/minute.

The accuracy of the flows obtained with the flow gauge was measured using a Brooks flowmeter for each individual graduation on the dial (Table 2). This shows the maximum error to be approximately 18%. However, most flow rates were more accurate than this, particularly at higher flows. The contents were assessed by weighing and by observing the contents gauge. In both respects the manufacturer's specifications (Table 3) were accurate to within 5%. The filling procedure will account for this error.

When not in use, the pressure in the container is controlled by venting excess gas to atmosphere via the relief valves. This limits the time for which liquid oxygen can be stored in the portable container. The rate of venting was measured by weighing (Fig. 3).

The system was easy to operate. Although there is a potential hazard from spilling liquid oxygen, this did not

occur at any time. The venting valve is designed to remain closed for 60 seconds if the unit is left on its side. Under the conditions to which the units have been subjected, such an event has occurred on numerous occasions but no spillage has been noted.

The system has been used to power the Pneupac Transpac, the Ambu uni-suction apparatus and hollow fibre oxygenators for patients on extracorporeal membrane oxygenation.<sup>1</sup>

### Discussion

There is good evidence that long-term oxygen therapy improves the vital and functional prognosis of patients with hypoxic lung disease.<sup>1,2</sup> In the USA, a significant proportion of the patients requiring domiciliary oxygen use a liquid oxygen delivery source.

The light weight and portability of liquid oxygen has resulted in improved patient compliance particularly when compared with both cylinders and concentrators,<sup>3</sup> but its use in the UK has been impeded by the exclusion of liquid oxygen systems from the Drug Tariff.

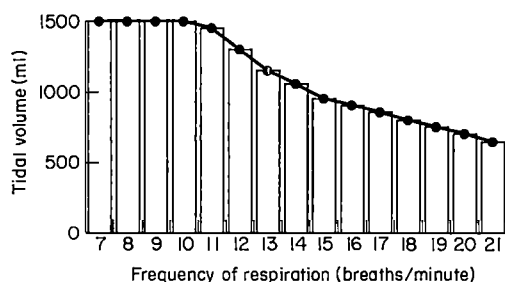


Fig. 2. Performance profile of the portable liquid oxygen unit operating at 350 kPa using a Pneupac ventilator. —●—, denotes the maximum attainable minute volume for the unit operating at this pressure.

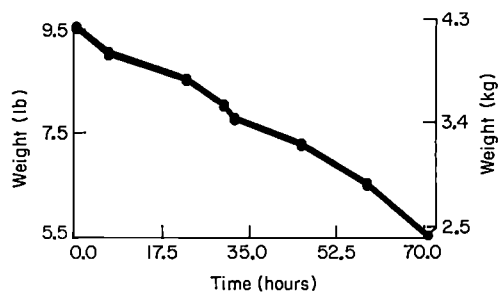


Fig. 3. Normal evaporation rate of oxygen from the portable liquid oxygen unit, measured by weighing.



**Table 1.** Performance profile of the Companion EMT operating at 350 kPa (A) and 450 kPa (B) using the Pneupac Transpac Ventilator.

Column 1			Column 2		Column 3	
Ventilator setting			Delivered tidal volume A (ml)	(% error)	Delivered tidal volume B (ml)	(% error)
Minute volume (litres)	Set rate (per minute)	Expected tidal volume (ml)				
20	40	500	450	(10.0)	500	(0)
20	30	667	600	(10.0)	700	(5.0)
20	20	1000	400	(60.0)	1150	(15.0)
20	10	2000	650	(67.5)	1750	(12.5)
18	10	1800	650	(64.0)	1575	(12.5)
14	10	1400	1100	(21.0)	1400	(0)
10	10	1000	950	(5.0)	1000	(0)

**Table 2.** Performance profile of the flow gauge.

Nominal setting (litres/minute)	Measured range (litres/minute)	Maximum % error
1.0	0.85– 1.15	15.0
1.5	1.25– 1.75	17.0
2.0	1.68– 2.36	18.0
3.0	2.62– 3.44	15.0
4.0	3.50– 4.55	14.0
5.0	4.40– 5.70	14.0
6.0	5.30– 6.85	14.0
8.0	7.00– 9.00	12.5
10.0	8.75–11.25	12.5
15.0	13.25–17.00	13.0

Liquid oxygen offers practical advantages in hospital for mobilising patients.<sup>4</sup> However, previous systems have been unable to provide oxygen at high pressure. This has precluded their use with gas-driven ventilators. The present system offers a significantly smaller and lighter unit compared with oxygen cylinders and contains the equivalent of 1.5 size E cylinders. It is also able to supply 40 litres/minute of oxygen at 450 kPa and thus can power gas-driven medical devices.

Liquid oxygen may also be useful when anaesthesia is administered outside hospital, for example, by the armed forces.<sup>5</sup> The fact that it is portable, and is a relatively low pressure source compared with oxygen cylinders, adds to its safety and convenience. In a military setting it may also be used to power gas-driven equipment, or as the oxygen source during drawover anaesthesia.

The system described in this paper is likely to undergo further modifications before it becomes widely available.

The problem of isolating the flow-controlled oxygen from the high pressure oxygen needs to be addressed in particular. Nevertheless, under circumstances in which size and weight are at a premium, this system using liquid oxygen has significant advantages over cylinders.

#### Acknowledgments

The authors thank the Puritan-Bennett Corporation for their support and cooperation in the preparation of this manuscript. In particular, we acknowledge Mr D. Scott (Marketing Manager) and Mr J. Hayes (Vice President of the Cryogenics Division). Finally, we thank Drs M. Boscoe and D. Royston (Consultant Anaesthetists, Harefield Hospital) for their help in the preparation of the diagrams.

#### References

1. KEE SS, SEDGWICK J, BRISTOW A. Case report: Interhospital transfer of a patient on extracorporeal carbon dioxide removal. *British Journal of Anaesthesia*. (in press)
2. MRC WORKING PARTY. Long term domiciliary oxygen therapy in chronic hypoxic cor pulmonale complicating chronic bronchitis and emphysema. *Lancet* 1981; **i**: 681–6.
3. NOCTURNAL OXYGEN THERAPY TRIAL GROUP. Continuous or nocturnal oxygen therapy in hypoxemic chronic obstructive lung disease: A clinical trial. *Annals of Internal Medicine* 1980; **93**: 391–8.
4. VERGERET J, TUNON DE LARA M, DOUVIER JJ, FREOUR P, CARDINAUD JP, COURTY G, TAYTARD A. Compliance of COPD patients with long term oxygen therapy. *European Journal of Respiratory Disease* 1986; **69** (Suppl. **146**): 421–5.
5. BULL PT, MERRILL SB, MOODY RA, BAKER DJ, YATES A, GERAGHTY F, TIGHE SQM, SQUIRES SJ, STOOT CJ. Anaesthesia during the Falklands campaign. The experience of the Royal Navy. *Anaesthesia* 1983; **38**: 770–5.

**Table 3.** Manufacturer's specifications.

Measurement	Portable unit		Stationary unit	
Liquid oxygen capacity (litres)	1.19	21.0	31.0	41.0
Gaseous equivalent (1000 litres)	1.00	17.3	25.5	33.8
Height (cm)	35.05	67.3	81.2	96
Diameter (cm)	—	36.3	36.3	36.3
Empty weight (kg)	2.49	18.9	22.5	27
Full weight (kg)	4.27	41.4	56.2	72
Normal evaporation rate (kg/day)	0.45	0.72	0.72	0.72

## Cyclopropane and the Datex Capnomac

### Effect of cyclopropane on the single wavelength infrared measurement of volatile anaesthetic agents

D. G. MASON AND A. R. LLOYD-THOMAS

#### Summary

*We report the effect of cyclopropane used for induction of anaesthesia in children on the subsequent measurement of maintenance volatile anaesthetic agents with the single wavelength infrared absorption technique. After using cyclopropane to induce anaesthesia we have observed that falsely high readings of the expired maintenance agent occur for up to 60 minutes when using the Datex Capnomac. This is because of the effect of low concentrations of cyclopropane expired from the patient.*

#### Key words

*Anaesthesia; cyclopropane.  
Equipment; Datex Capnomac.*

The Datex Capnomac uses the infrared absorption technique to measure the inspired and expired percentage concentrations of either halothane, enflurane or isoflurane.<sup>1</sup> The Datex analyser uses the difference in absorption at the wavelength width of 3.3–3.5  $\mu\text{m}$  to distinguish between each volatile agent. The gain on the instrument is altered for each agent and as a result it can only measure one agent at a time. We have made some preliminary investigations into the effect of cyclopropane on the infrared analyser and the duration of washout of cyclopropane following a brief period of administration in children.

#### Method

A Wosthoff pump was used to present known concentrations of cyclopropane in nitrogen to the Datex multigas analyser. All the apparatus used was placed inside a fume cupboard and the cyclopropane flow was limited to 200 ml/minute. The analyser was first calibrated using the Datex 'Quick Cal' canister set for enflurane and then calibrated to zero in 100% nitrogen on the isoflurane setting. A single measurement was made at 0.5, 1.0 and 1.5% cyclopropane after both the analogue and digital outputs had stabilised.

#### Results

The results were recorded on a pen chart recorder via the analogue output (Table 1). From these data it is possible to

see (Table 2) that it only requires small concentrations of cyclopropane (especially with the halothane setting) significantly to interfere with anaesthetic gas measurement.

We have further investigated the significance of these findings in clinical practice. Five children (aged 12–18 months) undergoing surgery for repair of hypospadias were studied. Anaesthesia was induced with 50% cyclopropane in oxygen for 90 seconds and maintained by nitrous oxide 70% in oxygen by controlled ventilation with concurrent caudal extradural analgesia.

We found that it took up to 60 minutes (Table 3) on the halothane setting of the Datex Capnomac to reach < 0.1%

**Table 1.** Measurements obtained from the Datex multigas analyser presented with cyclopropane (isoflurane setting).

% Cyclopropane in N <sub>2</sub>	% reading
0.5	1.1
1.0	2.2
1.5	3.4

**Table 2.** Recalibrations for each agent setting in the presence of cyclopropane.

Agent setting	% cyclopropane
1% isoflurane	0.46
1% enflurane	0.37
1% halothane	0.07

D.G. Mason, MB, BS, DA, FFARCS, Senior Registrar, Nuffield Department of Anaesthetics, The John Radcliffe Hospital, Headington, Oxford OX3 9DU, A.R. Lloyd-Thomas, MB, BS, FFARCS, Consultant, Department of Anaesthesia, Hospital for Sick Children, Great Ormond Street, London WC1N 3JH.

Correspondence should be addressed to Dr Lloyd-Thomas please.

Accepted 3 September 1990.

**Table 3.** Measured washout times for cyclopropane using the Datex Capnomac.

Patients	Time (minutes) to < 0.1 % digital end tidal reading	
	Halothane	Isoflurane
1	60	40
2	60	35
3	55	35
4	45	30
5	55	35

end-tidal reading despite no further administration of volatile agent.

### Discussion

The inspired and expired measurement of the commonly used volatile inhalational anaesthetic agents is now

becoming an essential part of monitoring during anaesthesia. The infrared absorption technique is used by several of the commercially available analysers. From our investigations we have shown that the use of cyclopropane for induction of anaesthesia in children will falsely elevate the end-tidal measurements of subsequent volatile anaesthetic maintenance agents when using the Datex Capnomac. This is because of the excretion of low concentrations of cyclopropane. The use of a different wavelength band or the use of two separate wavelengths may overcome this anomaly. The use of single wavelength infrared absorption analysis for the measurement of volatile anaesthetic agents will give inaccurate readings if used after a cyclopropane induction.

### Acknowledgments

The Authors thank E. Palayiwa, Senior Physicist, Nuffield Department of Anaesthetics for her technical assistance.

## A coaxial technique for facilitating one-lung ventilation

I. D. CONACHER

### Summary

*A tube for bronchial intubation is described. A long (48 cm), small bore (5.0 mm internal diameter), cuffed, bronchial plastic tube is inserted coaxially within a large bore tracheal tube (10.0 mm) used for ventilation. The inner tube is designed primarily as a blocker to be inserted with a fiberoptic or optical bronchoscope, but can be effected blindly with a stylet. Several methods of inserting the inner tube and ensuring correct placement were used in 10 males undergoing thoracic surgery. If the bronchial cuff is inflated the tube can be used either as a blocker or as a conduit for suction and conventional and differential ventilatory techniques. Early clinical experience suggests that the technique is an alternative method of facilitating one-lung ventilation.*

### Key words

*Equipment; bronchial intubation.*

*Anaesthetic techniques; one-lung ventilation.*

One-lung ventilation (OLV) facilitates thoracic surgery. The anaesthetic techniques that have evolved to achieve this since the late Sir Ivan Magill established the principles of bronchial tube placement under direct vision<sup>1</sup> more than half a century ago, include the use of blockers, bronchial tubes and double-lumen tubes (DLT). As a result of developments in materials and optics, the use of plastic tubes and fiberoptic technology are tending to supplant classical techniques of establishing conditions for OLV. More recently a coaxial system, in which a modified bronchial tube is placed within a tracheal tube, has been advocated.<sup>2,3</sup> A simplified concept based on similar principles but in which the emphasis is on the inner tube being used in a blocking mode is reported. Conventional OLV is conducted through the outer, tracheal tube.

### Methods

**The inner tube.** Five-millimetre bore prototype tubes of siliconised polyvinyl chloride were prepared (Fig. 1). Each tube has an outer diameter of 6.8 mm, is 48 cm long, with a 2-cm cuff 1 cm from the distal end. The distal end is rounded and smoothed but not bevelled. The low-profile, high-volume cuff can be filled to capacity with 3–5 ml of air. The inflating conduit for the cuff lies within the wall of the tube and exits 10 cm from its proximal end, to which a 15-mm male connector can be fitted. A 64-cm length plastic stylet is provided.

**Principle of use.** The bronchial tube is passed through a large bore tracheal tube fitted with a fenestrated connector. Conventional ventilation, either manually or with a ventilator, is conducted through the tracheal tube while the bronchial tube is inserted through the self-sealing port in the fenestrated connector. Several techniques to ensure endobronchial intubation and laterality can be used.

**Method A.** The plastic stylet can be positioned in the desired bronchus through a rigid, Negus-type bronchoscope. The stylet is threaded through the tracheal tube which is inserted with conventional laryngoscopy after the bronchoscope has been removed. The stylet is then the guide over which the bronchial tube is manoeuvred into place. Observation of ventilation and auscultation, when the bronchial cuff is inflated, confirms blockage. A 3.5-mm paediatric fiberoptic bronchoscope will pass inside the blocker and can be used to assess the position of the tube.

**Method B.** The complete system is assembled and inserted at laryngoscopy. The bronchial tube is then advanced blindly and manipulated into position. Bronchial cannulation is again confirmed by observation and auscultation after the cuff is inflated and the stylet removed.

**Method C.** The tracheal tube is inserted conventionally. The bronchial tube is mounted either on a fiberoptic bronchoscope (*Method C1*) or a rigid, endviewing, optical bronchoscope (e.g. Wolf 3.5 mm) (*Method C2*), inserted through the fenestrated connector and advanced into position under direct vision. The bronchial tube has to be cut almost to the length of the bronchoscope before *Method C2* can be used.

I.D. Conacher, MD, FFARCS, FRCP (Ed), Consultant, Departments of Anaesthetics, Freeman Hospital, Newcastle upon Tyne NE7 7DN.

Accepted 19 June 1990.

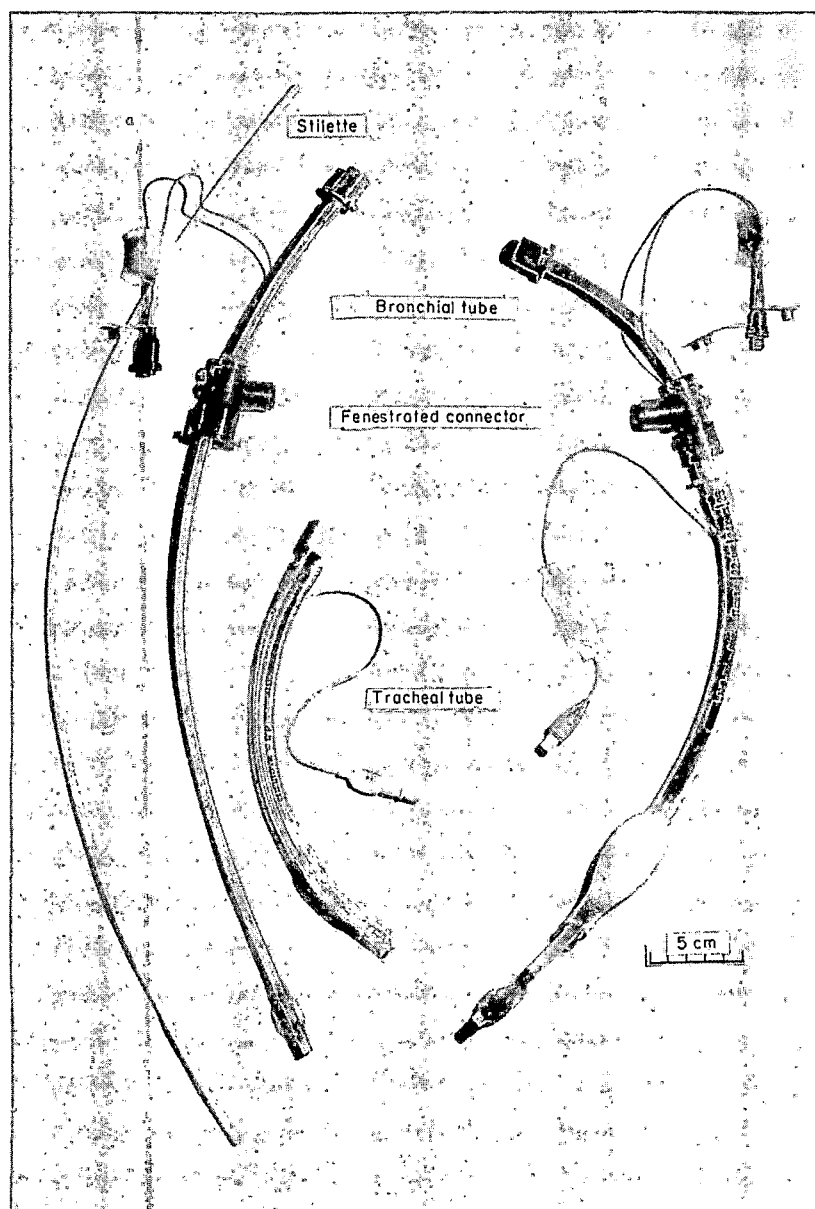


Fig. 1. The bronchial tube and intubation system prepared for intubation (left), and as utilised (right).

### Results

Bronchial cannulation proved relatively easy. The quickest, easiest and most convenient method of insertion under direct vision was with the rigid bronchoscope. The landmark for correct siting in the left main bronchus was the junction of the upper and lower lobes: that for siting in the *bronchus intermedius* on the right was sight of the carina between middle lobe and lower lobe.

The bronchial cuff was inflated with from 3–5 ml of air. In all cases this created an effective seal, although in three cases a leak on ventilation was still audible and palpable over the orifice of the bronchial tube at the ventilator end. Its existence did not appear to indicate either poor conditions for surgery or interference with dependent lung ventilation. On two occasions, when the cuff was overinflated, ventilator gauge pressure rose markedly. Both events occurred when the tube was in the right bronchial tree suggesting that cuff herniation might have interfered with ventilation down the tracheal tube.

*Early clinical experience.* The tubes were used on 10 males undergoing routine thoracic surgical operations through posterolateral thoracotomy incisions. Their ages ranged from 28–69 years, with a mean of 50.7 years. Weights ranged from 55–79 kg with a mean of 68.1 kg. Details are shown in Table 1. There were five left-sided main bronchus intubations and five right-sided. A size 10.0-mm tracheal tube was used as the main ventilatory conduit in all cases. In two of the cases the bronchial tube was inserted as designated by *Method A* above, three by *Method B*, three by *Method C1*, and two by *Method C2*.

In all cases the bronchial tubes were used primarily as blockers, but in several were also used in supplementary ways (Fig. 2). In three cases they were used for suction: in two, supplementary oxygen insufflation and in both of these some expiratory resistance (continuous positive expiratory pressure (CPAP)), was achieved with a PEEP valve attachment. In one case, a jet ventilation system was attached and the upper, surgical lung, ventilated differentially with intermittent high frequency ventilation.<sup>4</sup> In six

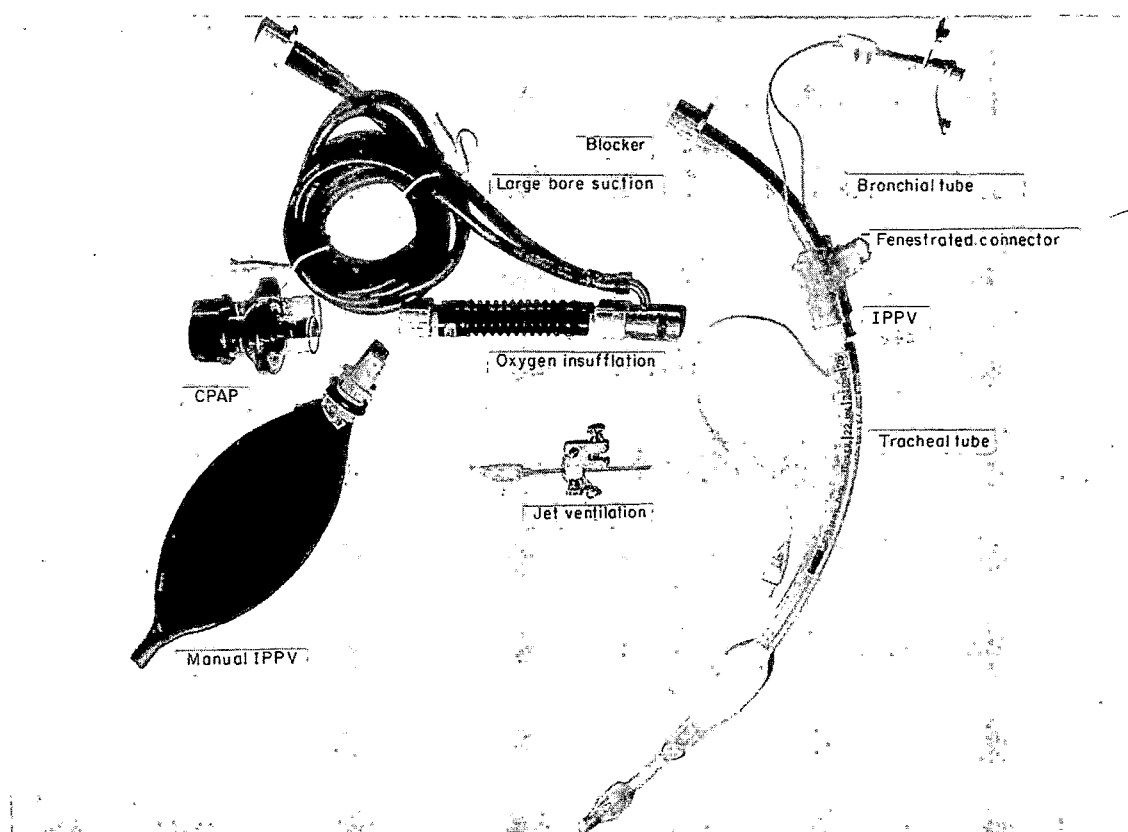


Fig. 2. Additional techniques employed with bronchial tube *in-situ*.

cases, the attachment of a hand ventilation set enabled reinflation of collapsed lung at the point of termination of OLV: in two of these it was necessary to deflate the bronchial cuff before upper lobe reinflation could be achieved. In one case the bronchial tube was removed before pneumonectomy; and in three before reinstitution of ventilation of both lungs. The arterial blood oxygen tensions ranged from 9.0–11.8 kPa with a mean of 10.0 kPa, during OLV with 30% oxygen in nitrous oxide, with normocapnia and without the addition of oxygen insufflation to the upper lung, or CPAP.

The bronchial tube obstructs ventilation on insertion through the tracheal tube, since it is advanced until the cuff is in the trachea and distal to the end of the tracheal tube. Formal bench testing of resistance to ventilation of the outer tube has not been carried out, but when the bronchial

tube is in position and used as a blocker (with cuff inflated and ventilator-end open to atmosphere) the pressure gauge on the automatic ventilator, connected to the tracheal tube, registered a peak inflation pressure similar to that seen when one lumen of a 39 FG DLT is ventilated.

In the cases where the right main bronchus was intubated the upper lobe bronchus was blocked. This did not appear to create a problem for the surgeons. If it was desired for the lobe to be actively collapsed (rather than by absorption collapse) then it could be emptied while ventilation was suspended with the bronchial cuff deflated.

### Discussion

Some essential characteristics of tried and tested techniques for facilitating OLV are safety, practicality, flexibility and

Table 1. Operation and intubation details. Ca, carcinoma.

Number	Diagnosis	Operation (side)	Intubation side and method (see text)	Comment
1	Empyema	Decortication (L)	Left A	Jet ventilation
2	Ca bronchus	Lower lobectomy (R)	Right B	
3	Ca bronchus	Wedge resection (L)	Left A	Position confirmed at fiberoptic bronchoscopy
4	Ca bronchus	Upper lobectomy (R)	Right B	Oxygen insufflation
5	Alveolar Ca	Pneumonectomy (L)	Left C1	Oxygen insufflation CPAP
6	Ca bronchus	Upper lobectomy (L)	Left B	Oxygen insufflation
7	Ca bronchus	Upper lobectomy (R)	Right C1	
8	Ca bronchus	Thoracotomy (R)	Right C1	
9	Pneumothorax	Pleurectomy (L)	Left C2	
10	Ca bronchus	Upper lobectomy (R)	Right C2	



simplicity. These are embodied in the described system.

The airway is protected from aspiration of material in the pharynx by utilising a technique that secures the upper trachea in the first instance; and patient oxygenation is ensured with the facility to apply ventilation continuously during manipulative manoeuvres.

Many of the objects used are available in anaesthetic rooms. Those that are not, but which are essential (e.g. fenestrated, self sealing connectors), are inexpensive. Others (e.g. optical and fiberoptic bronchoscopes), that make the system applicable to complex procedures are likely to be available in places that frequently use techniques of OLV.

An advantage of DLTs for thoracic surgery is flexibility. They are easy to insert and once sited enable collapse and (or) ventilation of either lung. Other techniques, notably the use of blockers, are not as utilitarian. The system described retains something of the flexibility, both as regards manoeuvres at will on individual lungs and suction of the intubated bronchus. Suction down the tracheal tube, however, is not possible when the bronchial tube is in position.

Blockage of an upper lobe bronchus, with the bronchial limb and blockage of a main bronchus, with a hernia of bronchial cuff, are associated with use of DLTs. Occurrence of such incidents is surmised from clinical observation, the sense of feel on manual inflation and auscultation. Similar events occur in practice with a coaxial technique but are more likely to be a problem when the bronchial tube is used for ventilation and not as a blocker. Cuff herniation is not detectable with a fiberoptic bronchoscope. There is insufficient space to pass a fiberoptic bronchoscope alongside the bronchial tube to view the cuff. However, against this disadvantage can be set the potential for reblocking a lung during surgery with a fiberoptic bronchoscope as a guide along which to manipulate the bronchial tube back into position should it slip out of a bronchus or be accidentally dislodged.

Nazari *et al.*<sup>3</sup> have enumerated the advantages of a coaxial system for facilitating one-lung anaesthesia and also suggested some indications other than thoracic surgery. They include a place in the assessment, management and therapy of unilateral pulmonary disease, lung

lavage and differential lung ventilation. They commend the safety features of having a wide-bore tracheal tube in place should problems occur and the ability to position the inner tube under direct vision. The resistance to ventilation is, however, considerable<sup>5</sup> and it remains to be seen if similar conditions pertain when, as in this report, the outer tube is the ventilation conduit.

The cost of disposable DLTs is approximately 10 times that of a tracheal tube, and to maintain a stock of sufficient range may involve considerable outlay. Customised right-sided versions are more expensive because of the extra cost involved in fashioning a ventilatory port for the upper lobe bronchus. The adoption of a simpler system with a single design suitable for either bronchus and utilising cheaper materials with a long shelf-life would probably result in significant cost saving for the occasional user of OLV techniques.

In preliminary trials the bronchial tubes have proved an alternative to other techniques of facilitating one-lung anaesthesia and ventilation.

#### Acknowledgments

Thanks are due to the Clinical and Product Research Department of Portex UK Ltd, Hythe, Kent, England for the prototype tubes used for this study.

#### References

1. MAGILL IW. Anaesthesia in thoracic surgery with special reference to lobectomy. *Proceedings of the Royal Society of Medicine* 1936; **29**: 643–53.
2. NAZARI S, TRAZZI R, MONCALVO F, ZONTA A, CAMPANI M. Selective bronchial intubation for one-lung anaesthesia in thoracic surgery. *Anaesthesia* 1986; **41**: 519–26.
3. NAZARI S, TRAZZI R, MONCALVO F, CARABELLA F, BELLINZONA G, BRASCHI A, SCARONI MT, MAPELLI A. A new method of separate lung ventilation. *Journal of Thoracic and Cardiovascular Surgery* 1998; **95**: 133–8.
4. PAES ML, CONACHER ID, SNELLGROVE TR. A ventilator for carbon dioxide laser bronchoscopy. *British Journal of Anaesthesia* 1986; **58**: 663–9.
5. CHIARANDA M, ROSSI A, MANANI G, PINAMONTI O, BRASCHI A. Measurement of flow-resistive properties of double-lumen tubes *in vitro*. *Anaesthesia* 1989; **44**: 335–40.

## Forum

### Wound infiltration of local anaesthetic after lower segment Caesarean section

T. N. Trotter, FCAnaes, Lecturer, P. Hayes-Gregson, FFA(SA), Senior Registrar, S. Robinson, BA, Research Sister, L. Cole, FFARCS, Lecturer, S. Coley, FFARCS, Senior Registrar, D. Fell, FFARCS, Senior Lecturer, University Department of Anaesthesia, Leicester Royal Infirmary, Leicester LE1 5WW

#### Summary

*The analgesic efficacy of subcutaneous wound infiltration with 20 ml of 0.5% bupivacaine after elective lower segment section Caesarean section was studied in 28 patients in a double-blind randomised controlled manner using a patient-controlled analgesia system. The mean 24-hour morphine consumption of the placebo group and the bupivacaine group was similar (76 mg and 68 mg respectively). Analysis of the cumulative hourly morphine consumption failed to show any statistically significant differences between the groups. However, on a weight-adjusted basis statistically significant differences in morphine consumption were demonstrated, although these may not be clinically important. Subjective experiences of pain, nausea and drowsiness assessed by linear analogue scoring were similar in both groups.*

#### Key words

*Pain, postoperative; patient controlled analgesia.  
Anaesthetics, local; bupivacaine.  
Anaesthetic techniques, regional; wound infiltration.*

The beneficial analgesic effect of subcutaneous wound edge infiltration using bupivacaine has been demonstrated following herniotomy in children and after excision of benign breast lumps.<sup>1,2</sup> However, the beneficial effects are less clear after upper abdominal surgery; reduction in opioid requirement after cholecystectomy has been demonstrated after infiltration of the peritoneum, muscle and subcutaneous tissues, but not after subcutaneous infiltration alone.<sup>3,4</sup> The opioid-sparing effect of subcutaneous wound infiltration with local anaesthetic agents has not been assessed previously after lower abdominal operations in adults, with the exception of herniorrhaphy. Patients undergoing Caesarean section mobilise early and would benefit from a technique which reduces opioid requirements and the incidence of related side-effects, and improves analgesia. Lower segment Caesarean section is performed routinely through a Pfannenstiel incision and is suitable, therefore, for the investigation of supplementary local anaesthetic techniques.

The purpose of this study was to assess the value of subcutaneous wound edge infiltration with bupivacaine after elective Caesarean section under general anaesthesia. Analgesia was assessed by reference to the use of intravenous boluses of morphine delivered by a patient-controlled analgesia system.

#### Method

Twenty-eight patients scheduled for elective Caesarean section under general anaesthesia were investigated in a double-blind, randomised trial. Each patient was visited pre-operatively and familiarised with the use of the PCAS. Patients gave informed consent and the study was approved by the District Ethics Committee. Patients were

not studied if they were unable to comprehend the nature of the assessments or the purpose of the PCAS, gave a history of sensitivity to opioids or local anaesthetics or had severe pre-eclampsia or severe hypertension of pregnancy. Premedication comprised ranitidine 150 mg orally administered on the evening before and on the morning of surgery and 30 ml sodium citrate 0.3 molar given orally immediately before induction of anaesthesia. Intravenous access was established and anaesthesia induced with a sleep dose of methohexitone followed by suxamethonium 1–1.5 mg/kg to facilitate tracheal intubation. Anaesthesia was maintained with 50% N<sub>2</sub>O in O<sub>2</sub> and supplemented with enflurane 1%. Muscle relaxation was accomplished with atracurium 0.5 mg/kg. The F<sub>IO<sub>2</sub></sub> at delivery was reduced to 0.3, and morphine 5–10 mg and syntocinon 10 IU were given. Atropine 1.2 mg and neostigmine 2.5 mg were administered at the conclusion of surgery to antagonise residual neuromuscular blockade.

Patients were randomly allocated between two groups to receive either 20 ml of bupivacaine 0.5% (group B) or a control group receiving 20 ml of normal saline (group C). The solutions were supplied by the hospital pharmacy in numbered vials in order to maintain blinding. After closure of the peritoneum the subcutaneous tissues were infiltrated with the trial solution by the surgeon, subject to a maximum of 0.4 ml/kg (2 mg/kg).

Postoperative analgesia was provided by a PCAS (Graseby Medical) connected by a Cardiff one-way valve to the intravenous infusion and provided morphine in 2-mg increments with a lockout time of 10 minutes. The number and timing of requests was recorded by an on-line printer (Hewlett-Packard 82162A Thermal Printer). Prochlorperazine 12.5 mg intramuscularly was given on request for nausea, and escape analgesia in the form of

Accepted 17 July 1990

intramuscular morphine was available to each patient. Patients returned to the ward after a short stay in the recovery area where they were reminded of the correct usage of the PCAS. Pain was assessed by the patient at 2-hourly intervals for the first 24 hours, utilising 10-cm linear analogue scales (LAS) for pain, in addition to ones for nausea and sedation. The assessment was omitted if the patient was asleep. Overall efficacy of analgesia was rated at the end of the 24-hour period using a four-point verbal rating score (VRS) comprising very good, good, moderate, or bad.

Systemic arterial pressure, heart rate and ventilatory rate was recorded by the ward nursing staff at regular intervals. The study was terminated at 24 hours, or earlier if mobilisation occurred spontaneously. Parametric data were analysed by Student's *t*-test; the Wilcoxon rank sum test was used for the data derived from LAS, and the Chi-squared test for the VRS. A *p* value of less than 0.05 was interpreted as an indication of statistical significance.

### Results

Fourteen patients received wound infiltration with bupivacaine (group B) and 14 (group C) received normal saline. There were no significant differences between the two groups with respect to age and height, but a significant difference emerged between the groups with respect to weight (Table 1). The patient weight was that recorded at the booking clinic at between 12 and 16 weeks' gestation. This was considered to be an adequate estimate of immediate postdelivery weight. There were no patients with multiple pregnancies or pathological causes of excessive weight gain in either group.

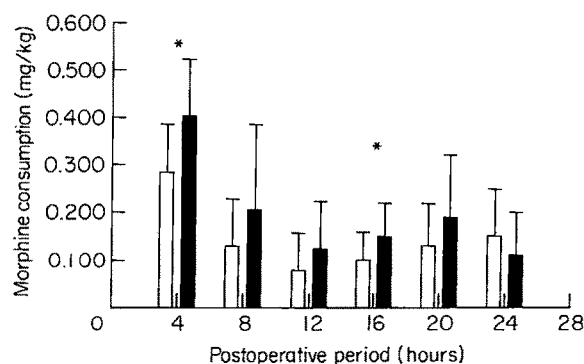
No problems were encountered with the PCAS in relation to programming or function, but data were lost from two patients in group C when the printer failed to record patient demands and only the 24-hour morphine consumption could be ascertained. Two further patients, one from each group, terminated the study at 20 and 21 hours respectively because of early mobilisation and discontinuation of the intravenous analgesia. In these patients, only data from the first five intervals are included.

Morphine consumption was compared over each 4-hourly interval, over the first 12 hours and over the 24-hour study period. There were no significant differences between the groups over these intervals; the mean (SD) 24-hour consumption for group B being 68.6 (23.2) mg and for group C 76 (25.2) mg. Total morphine consumption varied widely over 24 hours (group B 30–114 mg, group C 44–120 mg). Mean morphine consumption/kg body weight over the same intervals was calculated because of the disparity in patient weight. Patients in group B consumed significantly less over the first 4 and 12 hours and over the whole 24-hour period. Comparison of the remaining intervals showed no significant differences, with the exception of the period between 12 and 16 hours (Fig. 1 and Table 2).

Linear regression calculations were performed to assess correlation between weight and morphine consumption over 24 hours. For group B the correlation was 0.45 and for group C 0.39. Linear analogue scores for pain, nausea and sedation revealed no significant differences between the groups (Wilcoxon Rank Sum test). There was no clinical

**Table 1.** Patient data (mean, SD). \* *p* < 0.05 compared with group B.

	Group B	Group C
Age; years	31.6 (5.1)	28.7 (4.3)
Height; cm	157.6 (7.1)	158.7 (6.7)
Weight; kg	70.1 (13.7)	58.5 (14.9)*



**Fig. 1.** Four-hourly morphine consumption in the saline (■) (*n* = 14) and the bupivacaine (□) (*n* = 14) groups. Bars represent SD. \* *p* < 0.05 compared to saline.

evidence of excessive sedation or respiratory depression in any patient in the study.

Both groups expressed a high degree of satisfaction with the quality of analgesia provided; the verbal rating assessment of quality of analgesia showed no difference between groups (Table 3). The linear analogue scores for pain, nausea and sedation are shown in Table 4. There were no significant differences at any time.

Power analysis was carried out on weight-related and nonweight-related data from a nomogram described by Altman.<sup>5</sup> A 33% difference in opioid uptake was set as the minimum clinically important difference between the groups one would wish to detect, and is a similar difference to that demonstrated by others.<sup>3</sup> The number of patients was adequate to detect a difference of this magnitude with a power of 0.8 and *p* < 0.05 for the 12- and 24-hour periods, but for individual 4-hour intervals a larger study size would have been required.

### Discussion

This study demonstrates that subcutaneous wound infiltration with bupivacaine 0.5% did not decrease morphine requirements on the first postoperative day. However, if patient weight is taken into account, differences between the groups emerge over 12 hours, 24 hours and in two of the 4-hour periods. Examination of the remaining 4-hour periods demonstrated that there was a high probability of a type 2 error, due to the wide variation in opioid uptake; to achieve an adequate power, large numbers of patients

**Table 2.** Morphine consumption (mg/kg) expressed as mean (SD) during the first 12 and 24 hours of PCAS administration. Both these figures include the 5–10 mg loading dose administered in theatre. \**p* < 0.05 compared with group B. C, control group; B, bupivacaine group.

	Group B	Group C
12 hours	0.61 (0.15)	0.85 (0.28)*
24 hours	0.97 (0.35)	1.36 (0.43)*

**Table 3.** Patient assessment of quality of analgesia, (number of patients per category and percent). C, control group; B, bupivacaine group.

	Group B	Group C
Bad	0 (0%)	0 (0%)
Moderate	1 (7.1%)	1 (7.1%)
Good	5 (35.7%)	5 (35.7%)
Very good	8 (57.1%)	8 (57.1%)

Table 4. Median linear analogue scores (ranges) for pain, sedation and nausea. C, control group; B, bupivacaine group.

	Hours											
	2	4	6	8	10	12	14	16	18	20	22	24
Pain												
Group C	50 (3-80)	29 (3-65)	23 (0-60)	20 (0-80)	29 (0-54)	27 (0-66)	20 (0-52)	23 (0-76)	32 (0-68)	31 (0-77)	32 (0-72)	24 (0-60)
Group B	63 (0-100)	42 (2-84)	27 (2-72)	38 (0-74)	25 (0-50)	23 (0-54)	23 (0-70)	42 (0-82)	35 (0-72)	38 (2-65)	30 (0-90)	27 (0-92)
Nausea												
Group C	4 (0-82)	2 (0-79)	2 (0-90)	3 (0-70)	1 (0-50)	0 (0-78)	0 (0-10)	0 (0-19)	0 (0-14)	0 (0-16)	1 (0-87)	0 (0-56)
Group B	2 (0-72)	0 (0-42)	1 (0-90)	0 (0-89)	0 (0-90)	3 (0-62)	0 (0-25)	3 (0-34)	0 (0-25)	3 (0-18)	0 (0-92)	0 (0-93)
Sedation												
Group C	49 (3-88)	48 (3-96)	51 (0-96)	46 (20-77)	28 (0-75)	33 (0-80)	33 (0-80)	36 (0-80)	25 (2-75)	34 (0-70)	32 (0-52)	25 (0-92)
Group B	59 (3-94)	51 (0-97)	55 (4-95)	57 (0-98)	58 (0-96)	54 (0-100)	36 (0-98)	63 (0-92)	42 (0-87)	41 (0-79)	9 (0-100)	23 (0-98)

would have been required. It was believed that this was unnecessary since a significant difference had emerged over the first 12- and 24-hour period.

A large variation in opioid uptake from a PCAS has been shown.<sup>6,7</sup> Although the relation between weight and opioid uptake was of sufficient magnitude to produce statistically significant differences in this study, others have not found weight to correlate with opioid uptake from a PCAS in a nonpregnant population.<sup>6,7</sup> Weight gain in pregnancy arises partially as a result of an increase in extracellular fluid volume which can persist after delivery, so the volume of distribution of intravenous drugs may be altered.<sup>8</sup> This may account for the observed difference in this study between weight-normalised data. However, further evidence of morphine disposition in the puerperium is lacking and these results should be interpreted with caution.

The results of this study reaffirm the value of patient-controlled analgesia after Caesarean section, demonstrated elsewhere.<sup>9,10</sup> In these studies PCAS provided better analgesia and patient satisfaction when compared with intramuscular as required narcotics. In this study PCAS also provided a very high degree of patient satisfaction, with over 90% of patients describing their pain relief as either good or very good. This may have therapeutic implications.

Previous studies<sup>1-4</sup> have usually relied for assessment upon differences in intramuscular opioid given on request and differences in pain scores. However, numerous factors influence this regimen including inappropriate dosage, delays in drug administration and fear amongst staff of drug addiction.<sup>11</sup> Thus it is not possible accurately to assess the opioid-sparing potential of an analgesic technique, and comparison between studies is difficult. PCAS overcomes such problems and provides a more ideal way of assessing the need for postoperative analgesia<sup>6</sup> since patients titrate themselves to an acceptable level of comfort, unless side effects or inappropriate programming limit opioid uptake. PCAS has been used to investigate pain relief after techniques including epidural opioids<sup>12</sup> and nonsteroidal anti-inflammatory drugs;<sup>13</sup> it provides a more rigorous test of analgesic regimens than intramuscular administration.

Thus, there are no similar studies on wound infiltration after lower abdominal surgery using PCAS with which to compare our results. Comparisons with results after cholecystectomy are invalid, because this is an upper abdominal incision and because of differences in methodology. For example, infiltration of all cut surfaces after cholecystectomy with 40-50 ml of 0.5% bupivacaine, in addition to oesophagogastric decompression and immediate duodenal feeding, resulted in a significant reduction in postoperative opioid requirements.<sup>4</sup> Again, following cholecystectomy, 50 ml of 0.25% bupivacaine reduced opioid requirement given on request by approximately one third over the first 24 hours; however, no assessment of quality of analgesia or side effects was made in this study.<sup>3</sup>

Wound infiltration has been shown to provide equivalent postoperative analgesia to ilioinguinal nerve block in children after herniotomy.<sup>14</sup> Bilateral ilioinguinal nerve blockade<sup>15</sup> using 20 ml of 0.5% bupivacaine has been shown to reduce opioid requirements (intramuscular on request) and pain scores after lower segment Caesarean section. Bilateral ilioinguinal nerve block may be a more appropriate technique in this situation since wound infiltration with a similar dose of bupivacaine was ineffective in reducing opioid requirements.

#### Acknowledgments

We thank our anaesthetic, obstetric and midwifery colleagues at Leicester Royal Infirmary for their cooperation.

## References

1. FELL D, DERRINGTON MC, TAYLOR E, WANDLESS JG. Paediatric postoperative analgesia. A comparison between caudal block and wound infiltration of local anaesthetic. *Anaesthesia* 1988; **43**: 107-10.
2. OWEN H, GALLOWAY DJ, MITCHELL KG. Analgesia by wound infiltration after surgical excision of benign breast lumps. *Annals of the Royal College of Surgeons of England* 1985; **67**: 114-5.
3. PATEL JM, LANZAFAME RJ, WILLIAMS JS, MULLEN BV, HINSHAW JR. The effect of incisional infiltration of bupivacaine hydrochloride upon pulmonary functions, atelectasis and narcotic need following elective cholecystectomy. *Surgery, Gynaecology and Obstetrics* 1983; **157**: 338-40.
4. MOSS G, REGAL ME, LICHTIG L. Reducing postoperative pain, narcotics, and length of hospitalization. *Surgery* 1986; **99**: 206-10.
5. ALTMAN DG. Statistics and ethics in medical research. 3 How large a sample? *British Medical Journal* 1980; **281**: 1336-8.
6. TAMSEN A, HARTVIG B, DAHLSTROM B, LINDSTROM B, SON HOLMDAHL MH. Patient-controlled analgesic therapy in the early postoperative period. *Acta Anaesthesiologica Scandinavica* 1979; **23**: 462-70.
7. DAHLSTROM B, TAMSEN B, PAALZOW L, HARTVIG P. Patient-controlled analgesic therapy, Part IV: pharmacokinetics and analgesic plasma concentrations of morphine. *Clinical Pharmacokinetics* 1982; **7**: 266-79.
8. MOIR DD, THORBURN J. *Obstetric anaesthesia and analgesia*. London: Balliere Tindall, 1986.
9. EISENACH JC, GRICE SC, DEWAN DM. Patient-controlled analgesia following Cesarean section: a comparison with epidural and intramuscular narcotics. *Anesthesiology* 1988; **68**: 444-8.
10. RAYBURN WF, GERANIS BJ, RAMADEI CA, WOODS RE, PATIL KD. Patient-controlled analgesia for post-Cesarean section pain. *Obstetrics and Gynecology* 1988; **72**: 136-9.
11. SRIWATANAKUL K, WEIS OF, ALLOZA JL, KELVIE W, WEINTRAUB M, LASAGNA L. Analysis of narcotic analgesic usage in the treatment of postoperative pain. *Journal of the American Medical Association* 1983; **250**: 926-9.
12. MCQUAY HJ, BULLINGHAM RES, EVANS PJD, LLOYD JW, MOORE PA. Demand analgesia to assess pain relief from epidural opiates. *Lancet* 1980; **1**: 768-9.
13. COLQUHOUN AD, FELL D. Failure of rectal diclofenac to augment opioid analgesia after cholecystectomy. *Anaesthesia* 1989; **44**: 57-60.
14. REID MF, HARRIS R, PHILLIPS PD, BARKER I, PEREIRA NH, BENNETT NR. Day-case herniotomy in children. A comparison of ilio-inguinal nerve and wound infiltration for postoperative analgesia. *Anaesthesia* 1987; **42**: 658-61.
15. BUNTING P, MCCONACHIE I. Ilioinguinal nerve blockade for analgesia after Caesarean section. *British Journal of Anaesthesia* 1988; **61**: 773-5.

*Anaesthesia*, 1991, Volume 46, pages 407-409

### Midazolam-droperidol premedication for cardiac surgery

#### A comparison with papaveretum and hyoscine

J. H. L. Antrobus, BSc, MB, BS, FCAnaes, P. Abbott, MB, ChB, FFARCS, C. M. E. Carr, MB, BS, FFARCS, Senior House Officers, R. R. Chatrath, MB, BS, FFARCS, Consultant, Department of Anaesthesia, Killingbeck Hospital, York Road, Leeds 14.

#### Summary

*A combination of midazolam and droperidol given intramuscularly was compared with papaveretum and hyoscine for premedication of patients about to undergo cardiac surgery. Midazolam and droperidol proved to be a very satisfactory combination, producing superior sedation and anxiolysis with good cardiovascular stability.*

#### Key words

*Premedication; papaveretum, hyoscine, midazolam, droperidol.*

One of the principal functions of premedication is the relief of anxiety in the pre-operative period.<sup>1</sup> Anxiety is detrimental to morale and causes a tachycardia and elevation of arterial blood pressure which is undesirable in cardiac patients.<sup>2</sup> The combination of papaveretum and hyoscine continues to be used for premedication, although with the introduction of opioids in high dosage for induction of anaesthesia in cardiac surgery the analgesic effect of the premedication is not required, and the dry mouth caused by hyoscine is unpleasant for the patient. Despite their reputation for reliability, narcotic premedications still leave some patients relatively unsedated and anxious,<sup>3</sup> whilst the

maximum dose is limited by the central respiratory depressant effects.

Midazolam is a relatively new benzodiazepine which is anxiolytic, sedative, hypnotic, and causes anterograde amnesia.<sup>4</sup> Used intramuscularly it is reported to have a reliable effect as a premedicant.<sup>5-8</sup> Droperidol is a substituted butyrophenone which produces a state of calm and indifference with little hypnotic effect.<sup>9</sup>

This study was undertaken to compare the combination of midazolam and droperidol with papaveretum and hyoscine given intramuscularly for the premedication of cardiac surgical patients.

Correspondence should be addressed to Dr P. Abbott, Department of Anaesthesia, Royal Marsden Hospital, Fulham Road, London SW3 6JJ.

Accepted 12 September 1990.

**Table 1.** Changes in pulse rate (HR), systolic (SAP) and diastolic (DAP) pressures following premedication.

Group	n		Before premedication mean (SD)	Before induction mean (SD)
A	25	HR	74 (12)	71 (19)
		SAP	128 (14)	132 (18)
		DAP	77 (10)	81 (15)
B	25	HR	74 (13)	75 (13)
		SAP	129 (18)	118 (17)*
		DAP	77 (10)	76 (13)

\*Statistically significant from before premedication value,  $p < 0.05$ .

**Table 2.** Assessment of anxiety using visual analogue scores; mean (SD).

Group	Evening before	Before premedication	Before induction
A	3.3 (2.4)	3.2 (2.3)	2.6 (2.3)
B	3.4 (2.9)	3.6 (2.8)	1.9 (2.4)*

\* $p < 0.05$  compared to before premedication in group B.

### Methods

A double-blind, randomised, prospective trial was carried out on 50 adult patients who presented for elective cardiac surgery, either coronary artery bypass grafting or single valve replacement. Patients with Parkinson's disease and those taking regular psychotropic medication were not studied. All patients gave consent and the study was approved by the hospital ethics committee.

Patients were randomly allocated into two groups, group A receiving papaveretum and hyoscine and group B midazolam and droperidol one hour pre-operatively; the drugs were given intramuscularly. The groups were divided into three weight ranges, 40–55 kg, 55–70 kg and 70+ kg. Patients in the lower range received either 10 mg papaveretum with 0.2 mg hyoscine or 5 mg midazolam with 5 mg droperidol; those in the middle range either 15 mg with 0.3 mg or 7.5 mg with 7.5 mg and those in the upper weight range either 20 mg with 0.4 mg or 10 mg with 10 mg respectively. All received temazepam 20 mg on the evening before surgery and all were given supplemental oxygen via facemask following administration of the premedication.

Arterial pressure and pulse rate were measured immediately before premedication and again immediately before induction of anaesthesia after the insertion of peripheral venous and arterial lines under local anaesthesia.

An objective measure of anxiety was obtained using a visual analogue scale from 0 (I do not feel anxious at all) to

10 (the most anxious I have ever felt) on the eve of surgery, just before premedication and again before induction of anaesthesia.

Subjective assessment of sedation and of overall quality of premedication was made by an independent and highly experienced observer in the anaesthetic room who was not aware which premedication had been prescribed. Sedation was graded as wide awake, awake but drowsy, sleeping but easily rousable or sleeping and difficult to rouse. The overall quality of premedication was assessed as excellent, satisfactory or unsatisfactory.

The results were analysed using Student's *t*-test, Chi-squared or Wilcoxon's Rank sum test.

### Results

The mean (SD) ages and weights were group A, 53.2 (9.9) years and 78 (13.3) kg, and for group B, 54.6 (9.3) years and 71 (12.9) kg. These differences were not statistically significant.

**Cardiovascular.** The effects are shown in Table 1. In group A there were no statistically significant changes in systolic or diastolic arterial blood pressures or in pulse rate between baseline values and those immediately before induction. Group B showed no significant change in diastolic pressure or in pulse rate but the systolic pressure decreased from 129 (18.4) to 118 (16.9) mmHg following premedication ( $p < 0.05$ ).

**Anxiety.** The results are shown in Table 2. The mean anxiety visual analogue score decreased from 3.2 cm before premedication to 2.6 cm at induction in group A; this did not reach statistical significance. In group B the anxiety score decreased from a mean of 3.6 cm to 1.9 cm at these times, which was significant ( $p < 0.05$ ). However, when the two groups were compared with each other there were no significant differences between them at any assessment times.

**Sedation.** In group A, 14 patients were considered to be wide awake, 10 as awake but drowsy and one as asleep but easily rousable. No patients were difficult to rouse. No patients in group B were judged to be wide awake, 11 were awake but drowsy, 12 asleep but easily rousable and two were assessed as difficult to rouse. These differences between the groups were highly significant statistically ( $p < 0.001$ , Table 3).

**Overall quality of premedication.** Sixteen of group B patients were graded as excellent compared with four of group A. One patient in group B and none in group A were graded as unsatisfactory. The differences between the two groups were highly significant ( $p < 0.001$ , Table 3).

### Discussion

This study has shown the combination of midazolam with droperidol to be an effective premedication, in the dosage used, for adult patients undergoing cardiac surgery. In comparison with papaveretum and hyoscine it produced

**Table 3.** Assessment of degree of sedation and overall quality of premedication.

Group	Sedation on induction				Overall quality of premedication		
	Wide awake	Awake but drowsy	Sleeping easy to rouse	Sleeping difficult to rouse	Excellent	Satisfactory	Unsatisfactory
A, n = 25	14*	10	1	0	4*	21	0
B, n = 25	0	11	12	2	16	8	1

\* $p < 0.001$  statistically significant A from B.

greater sedation and higher overall quality of premedication. It is of interest that midazolam and droperidol, but not papaveretum and hyoscine, also significantly reduced the anxiety visual analogue scores since patients who appear well sedated may still express high levels of apprehension when asked to make a subjective assessment.<sup>3</sup>

Many cardiac anaesthetists prefer a heavy sedative premedication for patients undergoing open heart surgery;<sup>10</sup> the possible adverse effects of premedicant drugs on patients with poor cardiac function are compensated for by having a tranquil, sedated patient for induction of anaesthesia. Indeed, in some patients it is a definite advantage.<sup>2</sup> It may permit lower doses of induction agents and reduce left ventricular work by decreasing systemic vascular resistance. In patients with pulmonary oedema it may reduce hyperventilation and hence hypocapnia and vasoconstriction and by dilation of the venous capacitance vessels decrease pulmonary engorgement.

The midazolam-droperidol group showed a statistically significant decrease in systolic pressure from 129 to 118 mmHg. These patients were the more heavily sedated and a modest decrease of this magnitude is unlikely to be detrimental and may in fact be beneficial. Midazolam shows notable cardiovascular stability in patients with both ischaemic and valvular heart disease.<sup>11,12</sup> Droperidol causes no depression in myocardial contractility but does decrease both preload and afterload by alpha adrenoceptor blockade.<sup>13</sup> However, in ordinary clinical use it shows marked cardiovascular stability.<sup>9</sup>

Two patients, both of whom received the upper dose of midazolam and droperidol (approximately 0.11 and 0.12 mg/kg of each drug) were rated as 'difficult to rouse' in the anaesthetic room. One of these patients was subsequently graded as 'excellent' for overall quality of premedication and the other as 'unsatisfactory'. This anomaly represents a divergence of opinion between two independent and experienced cardiac anaesthetists on the merit of having very heavily sedated patients in the anaesthetic room.

Midazolam has been recommended for premedication at doses of 0.07 mg/kg, 0.08 mg/kg and 0.1 mg/kg,<sup>4-6</sup> although its efficacy in reducing subjective anxiety at 0.07 mg/kg has been questioned.<sup>14</sup> Doses of 0.15 mg/kg have been described as inducing 'rather deep sleep' whilst even higher doses may result in upper airway obstruction.<sup>15</sup> Patients in this study received between 0.1 and 0.14 mg/kg and although both drugs are known to be largely devoid of respiratory depressant properties in the usual dose range<sup>16,17</sup> care must be taken to adequately monitor them following administration of drugs intended to have a heavy sedative effect.

In conclusion, a combination of midazolam with droperidol provided a reliable method of producing the heavy preoperative sedation desirable for cardiac anaesthesia.

Papaveretum with hyoscine proved to be less reliable; the sedative and anxiolytic effect was limited since the dose cannot be greatly increased because of the risks of respiratory depression.

## References

1. NORRIS, W. The quantitative assessment of premedication. *British Journal of Anaesthesia* 1976; **41**: 778-84.
2. BRANTHWAITE MA. *Anaesthesia for cardiac surgery and allied procedures*. 2nd edn. Oxford: Blackwell Scientific, 1980.
3. FORREST WH, BROWN CR, BROWN BW. Subjective responses to six common preoperative medications. *Anesthesiology* 1977; **47**: 241-7.
4. DUNDEE JW, SAMUEL IO, TONER W, HOWARD PJ. Midazolam: a water-soluble benzodiazepine. Studies in volunteers. *Anaesthesia* 1980; **35**: 454-8.
5. MCATEER EJ, DIXON J, WHITWAM JG. Intramuscular midazolam. A comparison of midazolam with papaveretum and hyoscine for intramuscular premedication. *Anaesthesia* 1984; **39**: 1177-82.
6. FRAGEN RJ, FUNK DI, AVRAM MJ, COSTELLO C, DEBRUINE K. Midazolam versus hydroxyzine as intramuscular premedicants. *Anesthesiology* 1981; **55**: A278.
7. VINIK HR, REVES JG, WRIGHT D. Premedication with intramuscular midazolam: a prospective randomized double-blind controlled study. *Anesthesia and Analgesia* 1982; **61**: 933-7.
8. REVES JG, VINIK HR, WRIGHT D. Midazolam efficacy for intramuscular premedication: a double-blind placebo, hydroxyzine, controlled study. *Anesthesiology* 1982; **57**: A321.
9. VICKERS MD, SCHNIEDEN H, WOOD-SMITH FG. *Drugs in anaesthetic practice*, 6th edn. London: Butterworths, 1984.
10. LYONS SM, CLARKE RSJ, VULGARAKI K. The premedication of cardiac surgical patients. A clinical comparison of four regimes. *Anaesthesia* 1975; **30**: 459-70.
11. MARTY J, NITENBERG A, BLANCHET F, ZOUJIOUCHE S, DESMONTS JM. Effects of midazolam on the coronary circulation in patients with coronary artery disease. *Anesthesiology* 1986; **64**: 206-10.
12. SCHULTE-SASSE U, HESS W, TARNOW J. Haemodynamic responses to induction of anaesthesia using midazolam in cardiac surgical patients. *British Journal of Anaesthesia* 1982; **54**: 1053-7.
13. MARTY J, NITENBERG A, BLANCHET F, LAFFAY N, MAZZE RI, DESMONTS JM. Effects of droperidol on left ventricular performance in humans. *Anesthesiology* 1982; **57**: 22-5.
14. ARTRU AA, DHAMEE MS, SEIFEN AB, WRIGHT B. A re-evaluation of the anxiolytic properties of intramuscular midazolam. *Anaesthesia and Intensive Care* 1986; **14**: 152-7.
15. GEMPERLE M, KAPP W. Midazolam and anaesthesia. *British Journal of Clinical Pharmacology* 1983; **16**: 187S-90S.
16. POWER SJ, MORGAN M, CHAKRABARTI MK. Carbon dioxide response curves following midazolam and diazepam. *British Journal of Anaesthesia* 1983; **55**: 837-41.
17. PROKOCIMER P, DELAVAUULT E, REY F, LEFEVRE P, MAZZE RI, DESMONTS JM. Effects of droperidol on respiratory drive in humans. *Anesthesiology* 1983; **59**: 113-6.



## Patients' desire for information about anaesthesia

### Scottish and Canadian attitudes

M. Lonsdale, MB, ChB, FFARCS, G. L. Hutchison, MB, ChB, FFARCS, Senior Registrars, Department of Anaesthesia, Ninewells Hospital and Medical School, Dundee DD1 9SY.

#### Summary

*Patients in Canada and Scotland were asked to complete a pre-operative questionnaire examining their desire for information relating to anaesthesia. In both Canada and Scotland, patients under the age of 50 years had a greater wish to receive information than those who were older ( $p < 0.0001$ ). In Canada, female patients were found to be more keen to receive pre-operative information than males of the same age group ( $p < 0.05$ ). The priority given to individual pieces of information was remarkably similar in both countries. Details of dangerous complications of anaesthesia and surgery were consistently rated of low priority, with high priority going to postoperative landmarks such as eating and drinking. Both countries rated meeting the anaesthetist before surgery as the highest priority of all.*

#### Key words

*Anaesthesia.  
medicolegal; consent.*

There is wide variation in the policy of individual doctors regarding the quantity and quality of information they give to patients about their medical care. The reasons for this variation may include a reluctance to induce stress in patients by burdening them with unwanted information. Indeed Miller and Mangan, studying a group of American gynaecological patients, have shown that in some situations detailed information can cause increased tension, discomfort and depression.<sup>1</sup> It is essential to identify the aspects of peri-operative management that cause them most concern, before a coherent policy on dissemination of information to pre-operative patients can be developed. Anaesthetists visit their patients routinely before surgery. In this study we set out to identify what information patients want to be given by the anaesthetist about the peri-operative period. Also, in view of the increasing interest in the medicolegal implications of informed consent,<sup>2</sup> we examined two groups; a group of patients from Scotland, a relatively low litigation area, and a group from Canada, where litigation is more widespread.

#### Methods

Questionnaires were distributed in two locations: to patients attending for surgery in the Short Stay Unit, McMaster University Medical Centre, Hamilton, Ontario, Canada; and to general surgery inpatients in Ninewells Hospital, Dundee, Scotland. Both hospitals are teaching centres. The two towns are of comparable size and both based on manufacturing industry. Questionnaires were distributed pre-operatively to consecutive patients in each centre by an anaesthetist, who explained their purpose. They were completed without any supervision by medical or nursing staff.

Each questionnaire contained a list of 13 pieces of information relating to the experience of undergoing anaesthesia of any kind (Table 1). The patient was asked to tick a box indicating her/his feelings about receiving this information before anaesthesia. The available choices were 'prefer not

to know', 'Would like to know' and 'Feel I have a right to know'. In addition, a 14th section of the questionnaire asked for a response to the question, 'How do you feel about being visited before your operation by the person who will anaesthetise you?' The options given were analogous to those for the other 13 sections: 'Prefer not to meet them', 'Would like to meet them' and 'Have a right to meet them'. The patient's age, sex and number of previous operations were also recorded.

Scottish and Canadian questionnaires were identical, apart from minor linguistic differences such as the substitution of the term 'IVs' for the word 'drips' in the Canadian version. The data from the Scottish and Canadian studies were examined separately.

Table 1. The information section of the questionnaire.

1. Details of alternative methods of anaesthesia and their advantages and disadvantages.
2. Details of any drugs you will be given before your operation (to make you sleepy, etc.).
3. Where you will be anaesthetised.
4. Details of any needles, drips etc. used to give you your anaesthetic
5. How long you will be anaesthetised.
6. Where you will recover from your anaesthetic.
7. Whether you will have a drip, bladder catheter etc. when you wake up, and for how long.
8. What sort of pain you will have, for how long, and what sort of pain killers you can have.
9. When you will be allowed to eat and drink.
10. When you will be allowed to get up.
11. Details of all possible complications of anaesthesia and surgery.
12. Details only of dangerous complications.
13. Details only of common complications.

Patients were asked to choose from the options 'Prefer not to know', 'Would like to know' or 'Have a right to know' for each piece of information.

Firstly, the patients' desire for each of the 14 pieces of information was assessed. This was done by measuring the number of patients who responded to each question with one or both of the options 'Have a right to know' and 'Like to know'. These responses were to demonstrate a positive attitude towards receiving the particular piece of information concerned. The questions for both countries were ranked in order of the proportion of patients who gave such a positive response.

Secondly, both the Scottish and Canadian groups were divided into four age/sex subgroups: male < 50 years old, male  $\geq$  50 years old, female < 50 years old, female  $\geq$  50 years old. The total number of positive responses was calculated for each subgroup, as a measure of that group's overall desire for information. The groups were then compared to detect differences related to age or sex.

Finally, the data from each country were examined for the effect of the number of previous operations on the patient's overall desire for information.

Significance testing was performed using the Mantel-Haenszel test. This test is an extended form of the Chi-squared test, and is used when two separate factors, such as age and sex, may both influence an outcome simultaneously.<sup>3</sup> For example, it can be used to compare the two male and female age groups with each other, while ignoring any gender effects.

## Results

One hundred and thirty-eight patients over 18 years of age responded to the Canadian questionnaire (103 females). Their mean age was 45.2 (SD 19) years, with a mean 2.6 (SD 2.3) previous operations. All patients approached agreed to complete the questionnaire.

Forty-nine Scottish patients responded (22 females), with a mean age of 54 (14.3) years, and a mean 2.0 (1.7) previous operations. All patients asked agreed to complete the questionnaire, but one, a 74-year-old female, was subsequently unable to understand what was required.

The difference in age and sex distribution between the two countries was because of a preponderance of young female patients in the Canadian study. This is evident when the two nationalities are broken down into age/sex subgroups (Table 2).

**Table 2.** The age/sex subgroups examined. Number of patients per group, and the overall response in each category. ('Right to know' includes patients who ticked both 'Right to know' and 'Like to know')

	Scotland		Canada	
	M < 50 (n = 11)	M $\geq$ 50 (n = 16)	M < 50 (n = 18)	M $\geq$ 50 (n = 17)
Not to know (%)	18.83	21.88	10.71	15.97
Like to know (%)	58.44	41.96	63.49	58.40
Right to know (%)	15.58	4.02	22.62	15.55
No answer (%)	7.14	32.14	3.17	10.08
	Scotland		Canada	
	F < 50 (n = 10)	F $\geq$ 50 (n = 12)	F < 50 (n = 74)	F $\geq$ 50 (n = 29)
Not to know (%)	5.00	22.62	5.21	10.10
Like to know (%)	77.86	33.33	61.29	63.55
Right to know (%)	11.43	11.31	27.32	18.23
No answer (%)	5.71	32.74	6.18	8.13

F = female, M = male, < 50 = under 50 years old,  $\geq$  50 = 50 years old or more.

Nine of the Canadian patients (6%) and two Scots (4%) ticked both the options 'Like to know' and 'Have a right to know'. No other combinations were selected by any patient. Therefore, the grouping of these two options into the single category 'positive response' was adopted as an unambiguous indicator of a desire for information.

In both countries, the overall pattern of response was similar, but for each question, Canadians were more positive in their desire for information. Questions are arranged in order of positive response in Table 3. (When the rankings for the age/sex subgroups were examined, they did not differ in any major way from those obtained for the two countries overall.)

There were differences in attitude attributable to both age and sex. In Canada, females were significantly more positive in their desire for information than males ( $p < 0.05$ , Mantel-Haenszel test controlling for age), and younger patients more positive than older ones ( $p < 0.0001$ , Mantel-Haenszel test controlling for sex). In Scotland, the same age-related difference was found ( $p < 0.0001$ , Mantel-Haenszel test controlling for sex).

No significant effect attributable to the number of previous operations was detected in either Scotland or Canada. (The Mantel-Haenszel test was used to control for the effects of age, which might be covariable with the number of previous operations.)

## Discussion

Studies in Britain have shown that patients know little about their illnesses and the investigations they undergo<sup>4</sup> and are dissatisfied with the amount of information they

**Table 3.** Questions arranged in order of the proportion of patients showing a positive attitude ('Like to know' and/or 'Right to know').

Scotland		%
Information		
14. Meeting anaesthetist pre-operatively.		77
8. Pain/pain relief.		77
9. When allowed to eat and drink.		75
10. When allowed up.		75
7. Drips/catheters on recovery.		65
5. How long anaesthetised.		63
1. Alternative methods of anaesthesia.		61
2. Premedication drugs.		61
13. Common complications.		59
6. Location on recovery.		57
3. Where anaesthetised.		49
4. Needles, drips etc. used.		45
11. All complications.		43
12. Dangerous complications.		40
Canada		%
Information		
14. Meeting anaesthetist pre-operatively.		93
9. When allowed to eat and drink.		93
5. How long anaesthetised.		92
2. Premedication drugs.		90
10. When allowed up.		90
8. Pain/pain relief.		88
3. Where anaesthetised.		87
6. Location on recovery.		86
1. Alternative methods of anaesthesia.		85
13. Common complications.		85
7. IVs/catheters on recovery.		84
4. Needles/IVs etc. used.		80
11. All complications.		72
12. Dangerous complications.		66

receive from the medical profession.<sup>5</sup> Both Scots and Canadians, in this study, were positive in their overall desire for information, but the low scores found in Scottish patients reflect a significant number who selected 'Prefer not to know' in one or all of the sections.

The legal right of a patient to be given information differs widely between Britain and Canada. In Canada, where the doctrine of informed consent is applied, the patient decides personally what should happen to his/her body and has a legal right to a full explanation of all investigations and treatments, including all risks involved. In Britain, a doctor may decide on the appropriate treatment on the patient's behalf, without disclosing all the risks involved, so long as this management conforms to an accepted standard of medical care.<sup>6</sup>

It is impossible to guess the causal relationship (if any) between the medicolegal environment and patients' attitudes to information shown in this study. Legislation for informed consent may have occurred in Canada because patients wished it so, or Canadian patients may feel more strongly about receiving information because they are aware of a legal right to have it.

In both countries, patients under the age of 50 years were more positive in their desire for information, which supports the findings of Reynolds.<sup>5</sup> In Canada, females were found to feel significantly more positive about their desire for information than did males. One possible explanation for this finding is the publicity given at present to women's rights over their own bodies, as part of the abortion debate. A large amount of information on this topic is directed solely at a female audience; no corresponding issue exposes men, in quite such an immediate or emotional manner, to the same topic.

The pattern of response to individual questions was remarkably similar in the two countries, with both groups placing a similar emphasis on particular questions. In spite of the legal emphasis placed on disclosure of risk,<sup>7</sup> neither Canadian nor Scottish patients give a high priority to being informed about dangerous complications. This accords with the intuitive statements by many doctors that patients are reluctant to hear about the more serious complications of anaesthesia and surgery before operation. Both groups similarly placed a low priority on one of the other unpleasant aspects of anaesthesia: needles.

Moderate priority was assigned, in both countries, to a knowledge of common complications and to the topic of pain and analgesia. Over 20 years ago, Egbert *et al.* showed that patients given detailed information by their anaesthetist about pain and pain relief required less analgesia and were discharged from hospital sooner than less well-informed patients.<sup>8</sup> In this study, patients would appear to be at least receptive to similar initiatives.

The high priority placed on information about time to eating and drinking and mobility suggests that patients are most concerned about a clear timetable to recovery.

Meeting the anaesthetist before operation receives the highest score in both countries. Whether patients see this as a chance to obtain important information, or merely as an opportunity to assess the person to whom they are entrusting themselves, it behoves the anaesthetist, on either side of the Atlantic, to take note.

In conclusion, it would seem that patients do want to see their anaesthetist before operation and, regardless of medicolegal climate, have the same major concern: how soon they can anticipate a return to normal life. A comprehensive list of possible complications appears to be a very low priority. We agree with Miller and Mangan,<sup>1</sup> that detailed information about every aspect of medical care may cause some patients distress, and that further investigation is required to discover whether such information benefits or adversely affects the patients who receive it.

#### *Acknowledgments*

We are indebted to Dr J. B. Forrest and Ms J. Taylor, Department of Anaesthesia, McMaster University Medical Centre, and to the staff of the Short Stay Unit, McMaster University Medical Centre, for their assistance with the Canadian part of the study. Our thanks are also due to Mr S. Ogston, Department of Community and Occupational Medicine, Ninewells Hospital, for his advice on the statistical analysis of the data.

#### *References*

1. MILLER SM, MANGAN CE. Interacting effects of information and coping style in adapting to gynecologic stress: Should the doctor tell all? *Journal of Personality and Social Psychology* 1983; **45**: 223-36.
2. BYRNE DJ, NAPIER A, CUSCHIERI A. How informed is signed consent? *British Medical Journal* 1988; **296**: 839-40.
3. SCHEFLER WC. *Statistics for health care professionals*. Reading, Massachusetts: Addison-Wesley Publishing Co., 1984.
4. DUNKELMAN H. Patients' knowledge of their condition and treatment: How it might be improved. *British Medical Journal* 1979; **2**: 311-4.
5. REYNOLDS M. No news is bad news: Patients' views about communication in hospital. *British Medical Journal* 1978; **1**: 1673-6.
6. What should a doctor tell? News and Notes. *British Medical Journal* 1984; **289**: 325-6.
7. Sudaway v. the Bethlehem Royal Hospital and Maudsley Health Authority. *The Times* 1985 Feb 22.
8. EGBERT LD, BATTIT GE, WELCH CE, BARTLETT MK. Reduction of postoperative pain by encouragement and instruction of patients: A study of doctor-patient rapport. *New England Journal of Medicine* 1964; **270**: 825-7.

Flumazenil in intensive care

The duration of arousal after an assessment dose

G. C. Fisher, BM, FFARCS, Senior Registrar, Midlands Anaesthetic Training Scheme, M. C. C. Clapham, MB, BS, FFARCS, Consultant, Intensive Care Unit, East Birmingham Hospital, Birmingham, P. Hutton, BSc, PhD, MB, ChB, FFARCS, Professor, University of Birmingham, Clinical Research Block, Edgbaston, Birmingham.

Summary

The effect of an assessment dose of the benzodiazepine antagonist flumazenil was studied in 20 patients in an intensive care unit. The patients had been sedated with alfentanil and midazolam, and were ready to be weaned from mechanical ventilation. In 10 patients flumazenil was titrated just to produce full arousal whilst the midazolam infusion was continued; flumazenil administration was repeated one hour later after the infusion of midazolam had been stopped. In another 10 patients, flumazenil was administered only once, coinciding with the cessation of sedation. The duration of full arousal in both groups was less than 15 minutes in 75% of patients given a single dose of flumazenil (median dose 0.4 mg) although some effect persisted for up to 60 minutes. The cardiovascular effects of arousal were transient and probably not clinically significant. A brief duration of action is advantageous if the patient is found still to require sedation.

Key words

Antagonists, miscellaneous; benzodiazepines, flumazenil.  
Hypnotics, benzodiazepines; midazolam.

Hypnotics and opioids are used in the intensive care unit (ICU) to facilitate ventilation of the lungs and to ensure the patient's comfort.<sup>1</sup> Drugs given to depress airway reflexes, respiratory drive and conscious level inevitably complicate the assessment of both the patient's neurological status and ventilatory capacity during weaning. Flumazenil can reverse the benzodiazepine component of these effects. Its relatively short half-life becomes advantageous if the patient is found still to require sedation. The purposes of this study were to determine the duration of action of a dose of flumazenil which was just sufficient to produce full arousal in ventilated patients and to quantify the cardiovascular effects of sudden awakening in the presence of adequate analgesia.

Methods

Ethics committee approval was obtained to study 20 patients who required sedation to accept mechanical ventilation. Patients with known neurological disease and those who had previously received long-term benzodiazepine therapy were not studied, as were those with circulatory instability or multiple organ failure. Patients were included in the trial after being stabilised on the sedation scheme and when well enough for weaning to begin. All biochemical and haematological abnormalities were corrected as far as possible, and intermittent mandatory ventilation (IMV) was started. Blood gas analysis was used to confirm that  $P_{aCO_2}$  was in the range 4–5 kPa and that  $P_{aO_2}$  was above 12 kPa. Sufficient time was allowed for the influence of other intra-operative drugs to have worn off before the study started. Other drugs such as antibiotics, inotropic agents or diuretics were given as indicated. Fluid balance was judged on clinical grounds. Consent was obtained pre-operatively from the patients admitted electively into the ICU, but otherwise from the nearest relative.

Sedation and analgesia were achieved with intravenous infusions of midazolam and alfentanil. The benzodiazepine

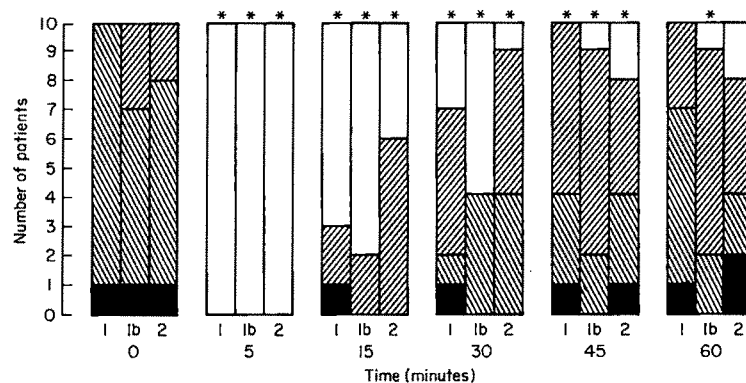
was titrated to produce a sedation score of 4 on a scale adapted from Ramsay *et al.*<sup>2</sup> (Table 1). The alfentanil infusion rate was based on previous publications<sup>3</sup> and our own clinical experience. Muscle relaxants were not used, and particular attention was paid to cardiovascular signs and purposeful movements suggestive of inadequate analgesia. Final adjustments were made to the infusion rates and the patients were observed for at least 2 hours to ensure stable conditions before the study commenced. Blood pressure was measured from an arterial line and the heart rate from the electrocardiograph monitor.

The 20 patients were allocated randomly to one of two groups.

*Group 1.* Blood pressure and heart rate were recorded each minute and the mean of five values taken as the baseline. Flumazenil was titrated intravenously in increments of 0.1 mg/minute until a sedation score of 2 had been achieved; the infusion rates of alfentanil and midazolam were not changed. The maximum heart rate and blood pressure observed during arousal were noted, as were the readings at 15, 30, 45 and 60 minutes afterwards. The midazolam infusion was then discontinued, the titration of

Table 1. Sedation score.

Score	Level of sedation/arousal
1	Anxious or restless.
2	Cooperative, accepting ventilation. Orientated and tranquil. Responds to commands.
3	Asleep. Brisk response to glabellar tap or loud auditory stimulus.
4	Asleep. Sluggish response to glabellar tap or loud auditory stimulus.
5	No response to glabellar tap or loud auditory stimulus, but responds to pain.
6	No response to pain.



**Fig. 1.** The effects on sedation of an assessment dose of flumazenil. Numbers of patients with each score before and 5, 15, 30, 45 and 60 minutes after titration. Flumazenil titrated during a midazolam infusion (group 1), second titration one hour later on stopping infusion (group 1b). First flumazenil titration on stopping midazolam infusion (group 2). Significant difference from baseline score denoted by \*  $p < 0.05$ , Sign test. ( $n = 10$ ). Key to shading; sedation score □ 2, ▨ 3, ▩ 4, ■ 5.

flumazenil repeated (group 1b) and the effects observed for a further 60 minutes.

**Group 2.** Stabilisation was achieved as described above. The midazolam infusion was stopped simultaneously with the start of flumazenil titration. The same schedule of observations was then applied.

Thus the changes in sedation due to flumazenil alone were studied by maintaining a constant infusion of midazolam during the observation period (group 1). Then combined effects of an antagonist and a declining level of agonist were assessed by first titrating the flumazenil immediately after stopping the midazolam infusion (group 1b), and secondly without the influence of a preceding dose (group 2). The alfentanil infusion was maintained unaltered throughout.

## Results

Twenty-two patients were entered into the study, but two subsequently met exclusion criteria and were withdrawn. One of these patients was confused on arousal and eventually was diagnosed as having viral encephalitis; the other developed septic shock during the observation period and required resuscitation. Most of the patients had received elective mechanical ventilation postoperatively, eight after aortic aneurysm repair, three after thoraco-abdominal oesophagectomy, and five after other extensive abdominal procedures; four patients had developed respiratory failure secondary to infection. Details of age and weight, sickness scores<sup>4</sup> and drug infusion data are shown in Table 2.

All patients were sedated satisfactorily using the combination of benzodiazepine and analgesic. The infusions

were adjusted to produce a sedation score of 4, although after the 2-hour period of observation two subjects were sedated more lightly and two more deeply than intended. Administration of flumazenil resulted in a score of 2 within one minute of the final intravenous increment. The dose required ranged from 0.2 to 1.0 mg; the median requirement was 0.4 mg. There were no differences in dosage among the three groups. None of the patients appeared anxious or agitated after arousal and no additional midazolam was required. The prompt return of the cough reflex was a feature of flumazenil-induced arousal and five patients no longer tolerated the tracheal tube; immediate extubation of the trachea was undertaken. The remainder continued to breathe via the IMV circuit or a T-piece.

The duration of flumazenil antagonism can be seen in Figure 1. The duration of full arousal was less than 15 minutes in 15 of the 20 patients after the first dose of flumazenil (groups 1 and 2) although a return to full sedation did not occur until 45 to 60 minutes after administration of flumazenil in those maintained on the midazolam infusion. The tendency to resedate was less pronounced when the midazolam had been discontinued and after a second flumazenil titration, although this difference was not statistically significant. Airway obstruction was an early feature of resedation in extubated patients who were left undisturbed.

The changes in heart rate and blood pressure were not significantly different among groups and therefore pooled data are shown in Table 3. Arousal was associated with transient increases in heart rate of 6 beats/minute and in systolic pressure of 14 mmHg.

Side effects attributable to flumazenil were minimal. One patient experienced nausea and one had an episode of facial flushing, which resembled the blush of embarrassment rather than that of histamine release. One subject had a brisk diuresis on arousal. No explanation for this could be found, and no changes in urine output were observed in the other subjects. One patient died from faecal peritonitis 5 days after the study.

## Discussion

The selection of patients in this study is biased towards those who received elective mechanical ventilation postoperatively because their consent could be obtained directly. The APACHE II scores are given because there appears to be a relationship between severity of illness<sup>5</sup> and sedation requirements. The scores are relatively low because, on the day of the study, the patients had recovered to the point of being weaned from ventilation, and because

**Table 2.** Patient and drug infusion data, as mean (range) unless otherwise stated.

Sex M/F	13/7
Age; years	64 (42–80)
Weight; kg	69 (47–93)
Median APACHE II score * on day of weaning	7 (0–12)
Alfentanil infusion rate; mg/hour	1.2 (0–3.5)
Duration of midazolam infusion; hours	48 (6–240)
Total dose of midazolam; mg	97 (30–800)
Midazolam infusion rate; mg/hour	2.9 (0.8–6.0)
Median dose of flumazenil; mg	
Group 1 First titration	0.4 (0.3–1.0)
Group 1 Second titration	0.4 (0.2–0.6)
Group 2	0.4 (0.2–1.0)

\* APACHE II score assuming a Glasgow Coma Score of 15.

**Table 3.** Systolic and diastolic blood pressures (mmHg) and heart rate (beats/minute) after flumazenil ( $n = 30$ ). Values expressed as mean (SD) (\* =  $p < 0.05$  compared to baseline).

	Baseline	Peak	15 minutes	30 minutes	45 minutes	60 minutes
Systolic	141 (31)	155 (39)*	151 (38)	146 (34)	148 (34)	148 (33)
Diastolic	71 (17)	78 (21)	71 (20)	68 (17)	71 (19)	68 (17)
Heart rate	87 (17)	93 (17)	90 (18)	88 (17)	89 (18)	87 (16)

all were given a Glasgow Coma Score of 15 despite being sedated. Oral tracheal tubes were used throughout the study although these might have been less well tolerated than nasal tubes. Intermittent mandatory ventilation was used routinely. At the time of the study, a score of 4 on the sedation scale was accepted as normal clinical practice and this was chosen as the end-point of midazolam titration. Alfentanil was chosen because its short half-life facilitates titration, its metabolites are inactive, and because it was thought to possess less sedative effect than some other analgesics. Alfentanil has been used extensively in ICU and, although drug metabolism can be disturbed in ill patients, we had not observed any specific cases of greatly extended duration of action.

The wide range of methods employed for sedation in the ICU indicates that the perfect regimen has not yet been developed. The combination of a benzodiazepine and an analgesic provides safe, good quality sedation<sup>6</sup> but accumulation remains a problem<sup>7</sup> and can complicate the assessment of a patient's cerebral and respiratory status. Even midazolam, the benzodiazepine with the shortest elimination half-life, may cause a prolonged period of sedation, possibly due to a pharmacogenetic subgroup of slow metabolisers,<sup>8</sup> or due to impairment of drug elimination in the severely ill.<sup>7</sup> Drugs with more rapid recovery characteristics have had their primary anaesthetic role extended to include sedation in the ICU, and isoflurane<sup>9</sup> and propofol<sup>10</sup> have been evaluated recently. However, experience with the benzodiazepines is considerable and now that the unpredictability of the recovery phase can be overcome with flumazenil, the physician has considerable control over the depth of sedation; full recovery can be achieved within seconds of an intravenous injection of flumazenil, and this is likely to be more rapid than waiting for the elimination of even a rapidly cleared drug.

A logical approach to evaluation of a patient's readiness to be weaned from mechanical ventilation is to use a small dose of flumazenil to assess the ability to breathe unaided and then to give additional doses if required. Flumazenil has a large therapeutic ratio and has been given safely to volunteers at doses much greater<sup>11</sup> than the currently recommended maximum dose of 2 mg. However, the use of a supramaximal dose to extend the duration of antagonism could be disadvantageous. A patient who is found to require continued mechanical ventilation, or who becomes excessively anxious on arousal,<sup>12</sup> is likely to be relatively resistant to further doses of a benzodiazepine agonist while an excess of antagonist remains at the receptor and other types of hypnotic may be needed to restore control. The more rapid re-sedation observed clinically in the patients in whom the midazolam infusion was continued (group 1) is evidence that sensitivity to the agonist drug was retained.

The effect of this small dose of flumazenil was demonstrable for over 45 minutes. However, we noted that airway obstruction was an early feature of re-sedation in the five patients in whom the trachea had been extubated. The loss of airway patency at such a light level of sedation may have been due to the combination of an airway desensitised by a prolonged period of tracheal intubation and the effects of alfentanil. There are obvious dangers associated with the

use of a short-acting antagonist to oppose the action of a longer-acting agonist if the physician is unaware of their respective kinetics. The patient must be observed for re-sedation until the agonist effects have worn off. This period of observation should continue for 2–4 hours after discontinuing a midazolam infusion,<sup>9,10</sup> but there is considerable individual variation. The patient must remain under close observation in the intensive care unit until both agonist and antagonist have declined to subtherapeutic levels.

The increases in heart rate and blood pressure on arousal were relatively transient, and only the changes in systolic pressure were statistically significant. Similar changes could have been expected during spontaneous reversal. In our previous study<sup>13</sup> of arousal from general anaesthesia with midazolam and alfentanil, there was little difference in this respect between control subjects and those in whom anaesthesia was reversed with flumazenil. Some cardiovascular response to arousal might be expected because intravenous midazolam can lower blood pressure<sup>14</sup> and flumazenil might be expected to reverse any receptor-mediated depression. The institution of positive pressure ventilation can reduce cardiac output, and reciprocal changes have been recorded during weaning.<sup>15</sup> In addition, it has been suggested that the ICU is a hostile environment in which to awaken suddenly.<sup>16</sup> Our study design may have minimised these changes by emphasising the adequacy of analgesia, the use of IMV and slow titration of flumazenil, but we suggest that patients who are particularly at risk from hypertension and tachycardia should be monitored carefully during arousal.

This study has confirmed that flumazenil is an effective antagonist of midazolam in the intensive care unit. Titration to an endpoint of full arousal allows assessment of a patient, and can be achieved with little circulatory disturbance. An awareness of its likely duration of action is essential for its safe use.

#### Acknowledgments

We thank our medical and nursing colleagues on the intensive care units of the Queen Elizabeth and East Birmingham hospitals for their cooperation, and Roche Products Limited for their financial assistance.

#### References

1. BION JF, LEDINGHAM IM. Sedation in intensive care — a postal survey. *Intensive Care Medicine* 1987; **13**: 215–6.
2. RAMSAY MAE, SAVEGE TM, SIMPSON BRJ, GOODWIN R. Controlled sedation with alphaxalone-alphadolone. *British Medical Journal* 1974; **ii**: 656–9.
3. O'DEA J, HOPKINSON RB. The combination alfentanil-midazolam by infusion: use for sedation in intensive therapy. *European Journal of Anaesthesiology* 1987; **1** (Suppl.): 67–70.
4. KNAUS WA, DRAPER EA, WAGNER DP, ZIMMERMAN JE. APACHE II: a severity of disease classification system. *Critical Care Medicine* 1985; **13**: 818–29.
5. BION JF, LOGAN BK, NEWMAN PM, BRODIE MJ, OLIVER JS, AITCHISON TC, LEDINGHAM I McC. Sedation in intensive care: morphine and renal function. *Intensive Care Medicine* 1986; **12**: 359–65.

6. WALLACE PGM, BION JF, LEDINGHAM I McA. The changing face of sedative practice. In: Ledingham IMcA, ed. *Recent advances in critical care medicine*, 3. Edinburgh: Churchill Livingstone, 1988.
7. SHELLY MP, MENDEL L, PARK GR. Failure of critically ill patients to metabolise midazolam. *Anaesthesia* 1987; **42**: 619-26.
8. DUNDEE JW, COLLIER PS, CARLISLE RTJ, HARPER KW. Prolonged midazolam elimination half-life. *British Journal of Clinical Pharmacology* 1986; **21**: 425-9.
9. KONG KL, WILLATTS SM, PRYS-ROBERTS C. Isoflurane compared with midazolam for sedation in the intensive care unit. *British Medical Journal* 1989; **298**: 1277-80.
10. AITKENHEAD AR, PEPPERMAN ML, WILLATTS SW, COATES PD, PARK GR, BODENHAM AR, COLLINS CH, SMITH MB, LEDINGHAM I McA, WALLACE PGM. Comparison of propofol and midazolam for sedation in critically ill patients. *Lancet* 1989; **2**: 704-9.
11. AMRIEN R. NDA Clinical expert report for Ro 15-1788 (Anexate). Roche 1986.
12. LOUIS M, FORSTER A, SUTER PM, GEMPERLE M. Clinical and hemodynamic effects of a specific benzodiazepine antagonist (Ro 15-1788) after open heart surgery. *Anesthesiology* 1984; **61**: A61.
13. FISHER GC, HUTTON P. Cardiovascular responses to flumazenil-induced arousal after arterial surgery. *Anaesthesia* 1989; **44**: 104-6.
14. REVES JG, FRAGEN RJ, VINK HR, GREENBLATT DJ. Midazolam: pharmacology and uses. *Anesthesiology* 1985; **62**: 310-24.
15. LEMAIRE F. Weaning from mechanical ventilation. In: Ledingham I McA, ed. *Recent advances in critical care medicine*, 3. Edinburgh: Churchill Livingstone, 1988.
16. RICOU B, FORSTER A, BRUCKNER A, CHASTONAY P, GEMPERLE M. Clinical evaluation of a specific benzodiazepine antagonist (Ro15-1788). Studies in elderly patients after regional anaesthesia under benzodiazepine sedation. *British Journal of Anaesthesia* 1986; **58**: 1005-11.



## Correspondence

<b>Studies on the laryngeal mask: first, learn the art</b>	417	<b>Modification of the laryngeal mask airway</b>	422
<i>A.I.J. Brain, FFARCS</i>		<i>M.Z. Michel, FCAnaes and M.M. Crosse, FFARCS</i>	
<b>Aspiration pneumonia and the laryngeal mask airway</b>	418	<b>Problems with the 32-gauge spinal catheter</b>	423
<i>P.L. Riddell, FFARCS</i>		<i>E.P. McCoy, FFARCSI and G. Furness, FFARCSI</i>	
<i>B. Philpott, FFARCS</i>	418	<b>Safe use of propofol in a child with acute</b>	
<i>A.I.J. Brain, FFARCS</i>	418	<b>intermittent porphyria</b>	423
<i>J.R. Krapez, FFARCS</i>	418	<i>A.S. Christian, FFARCS</i>	
<i>R.M. Griffin, FFARCS and I.S. Hatcher, MB, BS</i>	419	<b>Explanation for the overestimation of oxygen</b>	
<b>Aspiration and the laryngeal mask airway</b>	419	<b>saturation seen using nasal pulse oximetry</b>	424
<i>N. Koehli, FFARCS</i>		<i>I.J. Broome, FCAnaes and C.S. Reilly, FFARCS</i>	
<b>Son with lumière for cardiovascular monitoring</b>	420	<b>Modification of the Down's adjustable flow generator</b>	424
<i>A. Gilston, FFARCS</i>		<i>G.C. Fisher, FFARCS</i>	
<b>Midazolam withdrawal syndrome</b>	420	<b>Oesophageal rupture and cricoid pressure</b>	424
<i>I. McLellan, FFARCS and E. Douglas, RGN</i>		<i>W. Notcutt, FFARCS</i>	
<b>Failure of an Ohmeda Oxicap 4700</b>	420	<b>Bleeding postbronchoscopy</b>	425
<i>S.P.W. Spiers, FCAnaes and R.W. Harris, FCAnaes</i>		<i>C.A. Hodgson, FCAnaes</i>	
<b>'Finger-painting' and the pulse oximeter</b>	420	<b>Acute choreoathetoid reaction to propofol</b>	425
<i>J.R. Sneyd, FFARCS</i>		<i>P. McHugh, FFARCS</i>	
<b>Research difficulties for junior anaesthetists</b>	421	<b>Misdiagnosis caused by an epidural dressing</b>	425
<i>T.M. Akhtar, FFARCSI</i>		<i>A.M. Berry, FCAnaes and M.R. Patrick, FFARCS</i>	
<b>'Hazards' of oxygen therapy during spinal</b>		<b>The Woolley and Roe case</b>	426
<b>anaesthesia</b>	421	<i>J.R. Maltby, FFARCS</i>	
<i>R.H. Riley, MB, BS, K.A. Hammond, MB, BS and</i>		<b>Ketamine does not always work in the treatment of</b>	
<i>M.S. Currie, MB, BS</i>		<b>priapism</b>	426
<b>Angina haemorrhagica bullosa causing respiratory</b>		<i>B. Appadu, MD and I. Calder, FCAnaes</i>	
<b>obstruction postoperatively</b>	422	<b>Meningitis following spinal anaesthesia</b>	427
<i>S.I. Hosain, BM, G. Bounds, FRCS and J. Stanford, FFARCS</i>		<i>J. Van-Andel, MB, BS and N.G. Lavies, FFARCS</i>	

### Studies on the laryngeal mask: first, learn the art

Several correspondents have noted problems in the use of the laryngeal mask (LM) which have urged them to stress the need to avoid complacency.<sup>1-3</sup> Others' difficulties have prompted use of the superior laryngeal nerve block to overcome reaction to insertion<sup>4</sup> or suggest not using the device in those with hypersensitive respiratory reflexes.<sup>5</sup>

The instruction manual<sup>6</sup> lists a number of problems which may result from inadequate preparation or faulty insertion techniques. It is unfortunate that the best method of positioning the mask correctly, avoiding the epiglottis and the arytenoids during passage, is almost completely unknown, in spite of wide general use. Dr Nunn's group has shown radiographically that a number of important malpositions may occur without obvious clinical evidence.<sup>7</sup> All of these are shown in the manual, with instructions about how they can be avoided. Results<sup>1</sup> which indicate partial airway obstruction in 50% of children are surely cause for grave concern. The correctly positioned mask does push the arytenoids anteriorly, by an amount which probably

depends on the amount of air in the cuff,<sup>7</sup> but obstruction by the aryepiglottic folds<sup>2</sup> should not occur if the mask tip is correctly located against the upper oesophageal sphincter. If the mask tip is too high, which may occur if it is allowed to be dragged out of position by careless fixation for example, inflation of the cuff may result in the tip pushing the aryepiglottic folds anteriorly, thus causing obstruction.

Payne<sup>2</sup> mentions obtaining a fibrescopic view of the oesophagus through the mask bars: this can only be achieved if the mask has become folded over on itself, a consequence of poor insertion technique in which either lubrication has been inadequate or has been allowed to dry out, or the cuff has not been completely deflated or pressed up against the palate during insertion. It is important to note that fibrescopic views of the larynx are not three-dimensional. The aperture bars in the mask may appear to be completely obstructed by the partly downfolded epiglottis, while the patient continues to breathe normally (see the

All correspondence should be addressed to Dr M. Morgan, Editor of *Anaesthesia*, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, United Kingdom.

Letters must be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters contained in the Notice to Contributors to *Anaesthesia* printed at the back of each issue. The degrees and diplomas of each author must be given in a covering letter personally signed by all the authors.

Correspondence presented in any other style or format may be the subject of considerable delay and may be returned to the author for revision. If the letter comments on a published article in *Anaesthesia*, please send three copies; otherwise two copies of your letter will suffice.

instruction video). This is because gas is able to pass through the lateral spaces on either side of the bars. Breath sounds should be audible but quiet in the adequately anaesthetised patient. In children it is particularly important to rule out inadequate anaesthetic depth when breathing is noisy. If the mask is moved about in these circumstances, in the mistaken belief that it is in the wrong position, the result is likely to be complete obstruction due to laryngeal reaction. In children, again it is essential to use the recommended size. Recently, published data indicate that the size one is used in children between 13 and 15 kg,<sup>8</sup> over twice the recommended weight for size.

To use the laryngeal mask safely, it is important to carry out the following: (1) routine pre-operative testing for leaks or cuff herniation; (2) avoid excessive re-use (cuff herniation becomes more likely); (3) ensure the device is autoclaved before use (under no circumstances must glutaraldehyde, Cidex, be used); (4) correctly deflate and lubricate but avoid the anterior aspect of the mask; (5) pay scrupulous attention to correct insertion technique at an adequate depth of anaesthesia; (6) provide the recommended roll of gauze between the teeth to stabilise the position and prevent obstruction by biting. This must remain in place until the LM is removed; (7) avoid disturbing the device during anaesthesia or deflating the cuff until the patient's reflexes are fully returned; (8) ensure anaesthesia is adequate for the

level of surgical stimulus; (9) avoid rotation of the mask in the pharynx by bending the tube only downwards onto the chin, never upwards. Optimal stability can be achieved by passing the anaesthetic hosing under the pillow and connecting it to the LM tube below the chin.

Northwick Park Hospital,  
Harrow, Middlesex HA1 3UJ

A.I.J. BRAIN

#### References

1. ROWBOTTAM SJ, SIMPSON DL. Partial obstruction of the laryngeal mask airway. *Anaesthesia* 1990; **45**: 892.
2. PAYNE J. The use of the fiberoptic laryngoscope to confirm the position of the laryngeal mask. *Anaesthesia* 1989; **44**: 865.
3. WELSH BE. Will we ever learn? *Anaesthesia* 1990; **45**: 892.
4. DASEY N, MANSOUR N. Coughing and laryngospasm with the laryngeal mask. *Anaesthesia* 1989; **44**: 865.
5. BRAIN AIJ. The laryngeal mask in patients with chronic respiratory disease. *Anaesthesia* 1989; **44**: 790.
6. BRAIN AIJ. *The Intavent laryngeal mask instruction manual*. Henly-on-Thames: Intavent International SA, 1990: 43-53.
7. NANDI PR, NUNN JF, CHARLESWORTH CH, TAYLOR SJ. A radiological study of the laryngeal mask. *European Journal of Anaesthesia*: in press.
8. STONE PA, KILPATRICK AWA, THORNBURN J. The laryngeal mask airway in paediatric anaesthesia. *Anaesthesia* 1990; **45**: 924-7.

#### Aspiration pneumonia and the laryngeal mask airway

The case report by Drs Griffin and Hatcher (*Anaesthesia* 1990; **45**: 1039-40) on aspiration pneumonia and the laryngeal mask airway, was a salutary reminder of the inherent problems associated with this device.

Personally, I find it incredible that anyone would even consider using a laryngeal mask for cholecystectomy, where regurgitation of bile-stained gastric fluid is an all too frequent event, even under optimal conditions. I think the authors can consider themselves extremely fortunate that this is the only case of aspiration pneumonitis they have encountered if the laryngeal mask is being used routinely for upper abdominal surgery.

Selly Oak Hospital,  
Birmingham B29 6JD

P.L. RIDDELL

In Drs Griffin and Hatcher's case report (*Anaesthesia* 1990; **45**: 1039-40) they do not state whether operative cholangiography was performed. I suspect that it was. Many years ago, after this technique was introduced, I found an apparently high incidence of vomiting after gall bladder surgery. An investigation after operative cholangiography showed that the stomach contents were bile stained and radiographically opaque, demonstrating the passage from the duodenum to the stomach of the injected dye. It is possible that this retrograde passage may occur even without cholangiography, but I suspect that its incidence is less.

Whilst in no way detracting from the case report, this was a risk factor that was not discussed in the use of the laryngeal mask for this particular operative procedure and may not be as well known as perhaps it should be.

Royal Surrey County Hospital,  
Guildford, Surrey GU2 5XX

B. PHILPOTT

Drs Griffin and Hatcher (*Anaesthesia* 1990; **45**: 1039-40) have performed a valuable service in drawing attention to a fundamental requirement for safe use of the laryngeal mask

(LM), whether in spontaneous or controlled ventilation. The lesson of their paper is that the cuff must never be deflated until protective reflexes have fully returned, as judged by ability to open the mouth on command (see instruction manual, page 20, point 9, page 21, point 3, page 22, points 10 and 11). As their case illustrates, it is as dangerous to deflate the LM cuff prematurely as it would be to deflate the cuff of a tracheal tube at a similar stage of anaesthesia without performing suction. Suction is not usually required when using the LM, but this is only because it can be left in place until swallowing and cough reflexes are effective. To deflate and remove the LM before this stage is reached is to lose one of the cardinal advantages of the device and should be strongly discouraged.

Northwick Park Hospital,  
Harrow, Middlesex HA1 3UJ

A.I.J. BRAIN

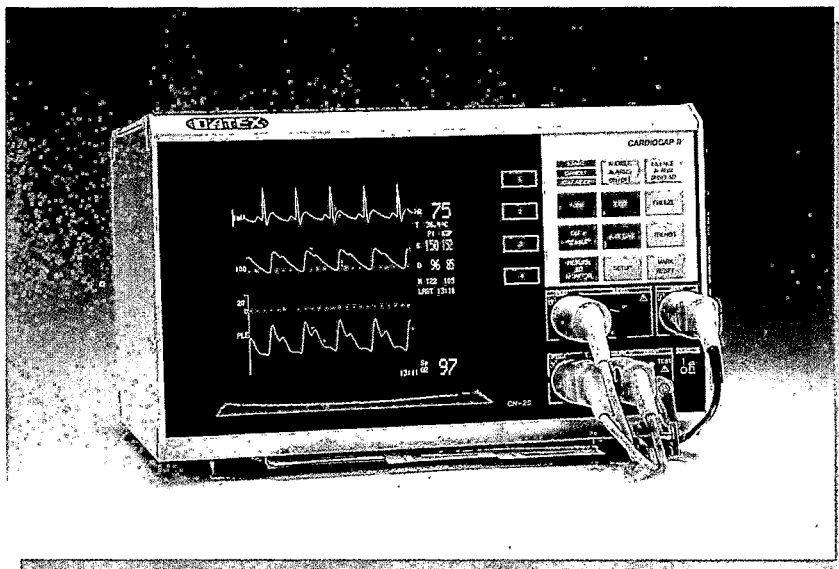
Drs Griffin and Hatcher are to be thanked for reminding us that the risk of aspiration pneumonitis is ever present during anaesthesia, including techniques which involve the use of the laryngeal mask airway (*Anaesthesia* 1990; **45**: 1039-340). May I comment on both the conclusions drawn by the authors and the use in general of the airway.

Patients with gall stones frequently have associated hiatus hernia. This patient would automatically be in a higher than average risk group for aspiration and it is questionable whether the use of the laryngeal mask was appropriate in this situation. There is in any case no need to postulate gastric distension caused by undetected gas leak, as suggested by the authors, although any such effect would clearly compound the underlying risk. I believe, however, that this report has highlighted a far more important consideration. I wish to suggest that the elective use of the laryngeal mask is wholly inappropriate in any patient who is paralysed and undergoing abdominal (or, for that matter, thoracic) surgery. Only tracheal intubation can provide the airway security and protection that is necessary

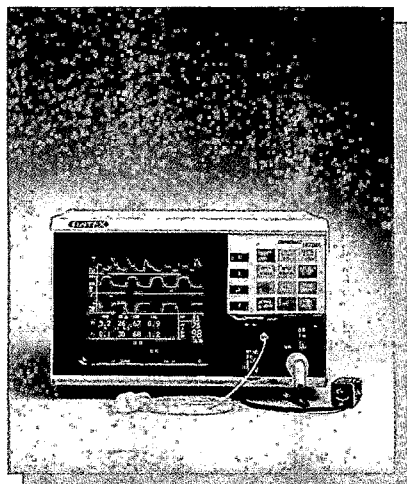
# Datex monitors : a reputation built on performance and reliability.

Datex has pioneered the development of modern airway gas monitoring ever since the introduction of their first compact capnometer in 1975.

Since then, Datex has played a major role in bringing CO<sub>2</sub> monitoring from a research curiosity to the standard of patient care in today's operating theatres.



*Cardiocap II - Employs the latest advances in non-invasive techniques for measuring blood pressure, oxygen saturation, peripheral pulse and airway gases. Available in 8 haemodynamic versions and 6 haemodynamic / gas versions.*



*Ultima - CO<sub>2</sub>, N<sub>2</sub>O, anaesthetic agent and patient O<sub>2</sub> monitoring as standard. Allows integration of SpO<sub>2</sub>, agent identification, airway pressure and tidal volume.*

Building on expertise acquired in CO<sub>2</sub> monitoring the technology was then extended to produce the *Normac* - the first anaesthetic agent monitor accurate and reliable enough for routine clinical use.

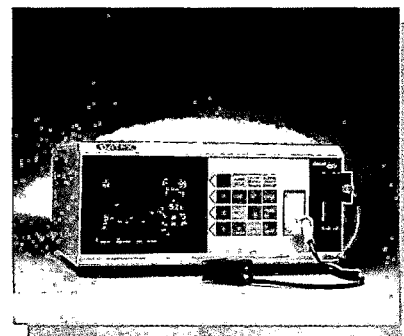
In 1985 the development of a solid state paramagnetic oxygen sensor was a significant breakthrough in clinical oxygen monitoring.

The new sensor required no maintenance since it had no chemical components, and its fast response time gave the added bonus of breath-by-breath measurement of expired and inspired O<sub>2</sub> concentrations.

Since then Datex have developed new monitors for measuring oxygen saturation, blood pressure and other physiological

parameters, devoting considerable resources to quality and reliability.

If you would like to try any of the Datex monitors then please contact S&W Vickers at any of the addresses below.



*Oscar-oxy - CO<sub>2</sub>, SpO<sub>2</sub>, N<sub>2</sub>O and patient oxygen all in a single, compact monitor. Ideal for bringing older theatres up to minimum monitoring standards.*

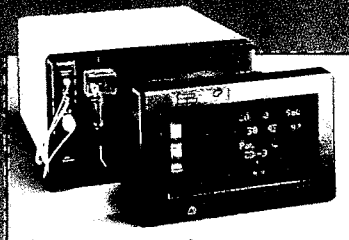
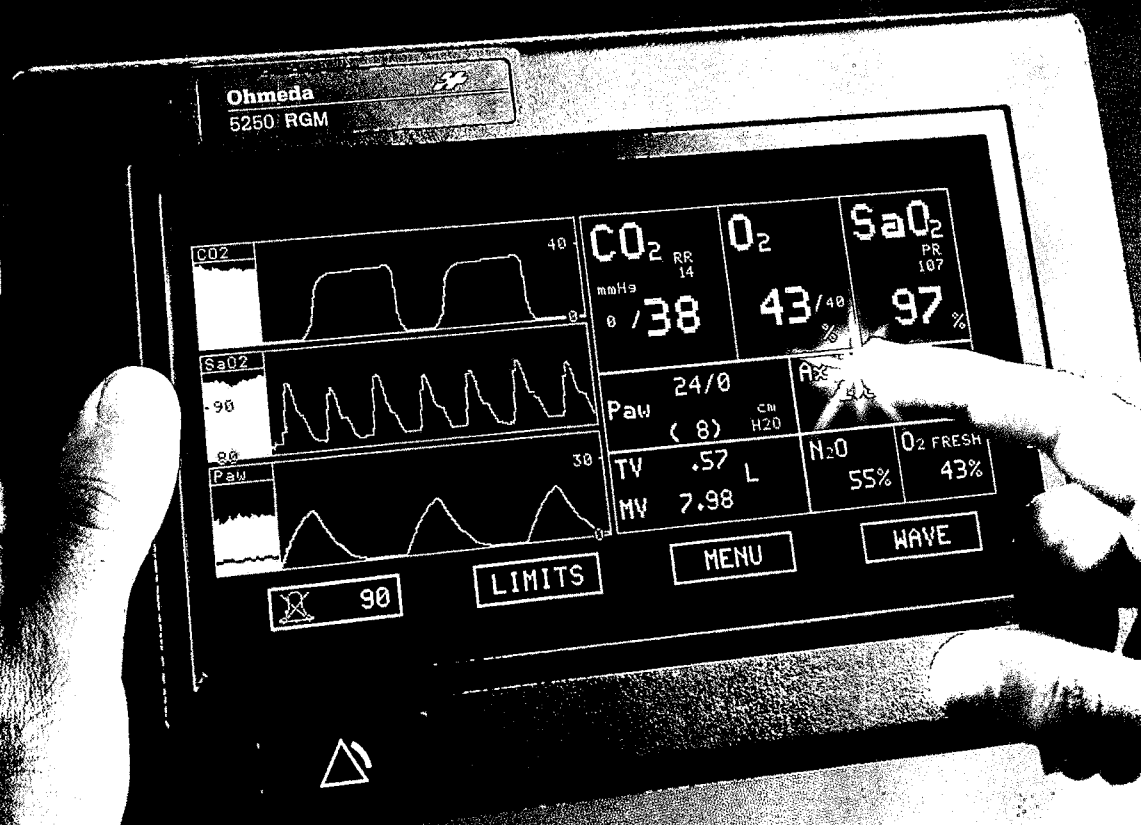
## S&W Vickers

32 Kerse Road, Stirling, FK7 7TA Tel 0786 51602 Fax 0786 51603

Ruxley Corner, Sidcup, Kent, DA14 5BL Tel 081- 309 0433 Fax 081- 309 0919

4 Balmoral Business Park, Boucher Crescent, Belfast, BT12 6HU Tel 0232 669266 Fax 0232 669223

# When you choose the RGM from Ohmeda, we really have to hand it to you.



Finally, total gas monitoring at your fingertips. Only the Ohmeda 5250 RGM combines up to seven parameters into one flexible system. With its removable touchscreen and easy to use menu, it puts the information you need right where you need it.

How does the RGM do it all? We really have to hand it to anesthesiologists like yourself. They wanted a simple, compact alternative to multiple monitors so they could watch digital, waveform, and trend displays—and their patients.

We've put today's most advanced monitoring technologies in your hands. Just what you'd expect from Ohmeda. Designing products with the clinician's needs in mind for over 80 years.

**Ohmeda**  
We monitor life.

71 Great North Road  
Hatfield  
Herts AL9 5EN  
United Kingdom  
☎ 0707 263570  
Telex UK 918179

**BOC**  
A BOC Health Care  
Company  
Critical Care  
Worldwide

A Division of The BOC Group Inc

© 1990 The BOC Group Inc

in these cases and any anaesthetist who fails to provide these safeguards would, in my opinion, be failing in his (her) duty to the patient.

I am a great believer in the laryngeal mask airway and would not wish to see it fall into disrepute as a result of inappropriate application. For all its drawbacks and complications, there are some situations where the tracheal tube is irreplaceable. I believe that this is one of them.

St Bartholomew's Hospital,  
West Smithfield, London EC1

J.R. KRAPEZ

#### A reply

Thank you for the opportunity to reply. The literature<sup>1</sup> and manufacturer's data sheet suggest that the laryngeal mask airway (LMA) can be used with intermittent positive pressure ventilation. However, this is not without risk of regurgitation of gastric contents and pulmonary aspiration, as demonstrated in our case report (*Anaesthesia* 1990; **45**:

1039–40). We accept there were factors, including the use of intra-operative cholangiography, in our patient which may have put her at greater risk of aspiration. Regurgitation happened on removal of the LMA, which may have occurred following extubation had a tracheal tube been used. Of greatest concern, which we reported, was the 'channelling' of the regurgitated material into the larynx by the LMA, which predisposed the patient to pulmonary aspiration.

As a consequence of our experience, we would not advocate use of the LMA for patients having major abdominal surgery. If it is used we would re-iterate the need for careful timing of cuff deflation and removal of the airway.

The Ipswich Hospital,  
Ipswich IP4 5PD

R.M. GRIFFIN  
I.S. HATCHER

#### Reference

1. BRAIN AIJ. Three cases of difficult intubation overcome by the laryngeal mask airway. *Anaesthesia* 1985; **40**: 353–5.

### Aspiration and the laryngeal mask airway

Drs Griffin and Hatcher (*Anaesthesia* 1990; **45**: 1039–40) recently reported a case of aspiration pneumonia during the use of a laryngeal mask airway (LMA). In this case aspiration occurred at the end of a 1.5-hour anaesthetic, during which positive pressure ventilation had been used. I would like to report another case of aspiration involving a laryngeal mask airway, but this time during the induction of anaesthesia.

A 51-year-old, 80-kg man with no relevant previous medical history was to be anaesthetised for insertion of a ureteric stent. Following pre-oxygenation, anaesthesia was induced with fentanyl 1.5 µg/kg and propofol 3 mg/kg. A size 4 LMA was inserted without difficulty and the cuff inflated with approximately 30 ml of air. Anaesthesia was maintained with 3% enflurane, 66% N<sub>2</sub>O and 33% O<sub>2</sub> by gentle manual ventilation of the lungs until spontaneous respiration returned. After three breaths the patient hiccupped and subsequently developed severe laryngeal spasm. Despite a smell of gastric contents nothing could be aspirated from the oropharynx. Cyanosis, of rapid onset, was noted and this was unresponsive to increasing the inspired oxygen concentration. Tracheal intubation, facilitated by suxamethonium, was performed and suctioning the trachea produced a small amount of clear fluid. The patient was transferred to the operating theatre, at which time the oxygen saturation, whilst breathing a fractional oxygen concentration (*F*O<sub>2</sub>) of 0.5, was 89%. At the same time it was evident that left chest movement was greatly diminished and breath sounds were absent on this side. The pulse rate and arterial blood pressure remained within acceptable limits. The operative procedure was rapidly completed and a chest X ray taken, which showed tracheal shift to the left along with left lower and midzone opacification. Fibreoptic bronchoscopy revealed normal right-sided bronchial appearance. On the left side the main bronchus was in severe spasm, which prevented further passage of the bronchoscope. No solid soiling was seen.

The patient was transferred to the ICU where controlled ventilation was continued. The *P*aO<sub>2</sub> at this time was 10 kPa with *F*O<sub>2</sub> 0.8. Treatment with regular nebulised salbutamol was started with a marked improvement in left-sided air entry and radiographic appearances. Sustained

improvement allowed the *F*O<sub>2</sub> to be steadily reduced and 20 hours later, immediately before extubation, the *P*aO<sub>2</sub> was 13 kPa whilst breathing 33% oxygen. The patient made an uneventful recovery after this.

Several previous letters to this journal (*Anaesthesia* 1990; **45**: 167–8) describe regurgitation of gastric contents into LMAs during emergence from anaesthesia. Regurgitation at this stage of anaesthesia is not an uncommon occurrence but is usually of little consequence because of positioning patients on their sides and returning laryngeal reflexes. Patients with LMAs remaining in place are often taken to recovery lying on their backs. This is because, firstly, the airway is well maintained in this position by the mask, secondly, the mask is probably felt to 'protect' the larynx and, thirdly, the stimulation of turning may result in premature rejection of the mask.

The above case provides evidence to support two elements of concern when using LMAs. Firstly, the lack of fluid present on oropharyngeal suctioning in this case seems to confirm the belief that, whilst the mask is in place with its cuff inflated, regurgitated fluid is prevented from escaping via the pharynx. This negates one advantage of the lateral position. Secondly, it must be assumed that in the 6% or so of cases in which the oesophagus is included within the aperture of the laryngeal mask,<sup>1</sup> the only thing that prevents regurgitated gastric fluid from being directed, preferentially, into the trachea is adequate laryngeal reflexes. This raises the question of whether LMAs really ought to be left in position for recovery staff to remove. Should they not be removed in the operating theatre and patients transferred in the lateral position?

Sir Humphry Davy  
Department of Anaesthesia,  
Bristol Royal Infirmary,  
Bristol BS2 8HW

N. KOEHLI

#### Reference

1. PAYNE J. The use of the fibreoptic laryngoscope to confirm the position of the laryngeal mask. *Anaesthesia* 1989; **44**: 865.

## Son with lumière for cardiovascular monitoring

We have reached the stage where real-time electronic monitoring during general anaesthesia has become virtually mandatory, and as with a ventilator alarm, a medicolegal requirement for even the simplest and shortest procedure.

There are still two desirable facilities which I have not seen on the equipment I know, and neither would be expensive to introduce or add to existing devices. Firstly, it should be possible to obtain a distinctive audible signal on a combined ECG/pulse oximeter for either variable because of the vital, although often overlooked, distinction between the electrical and the mechanical activity of the heart.<sup>1</sup> Cardiac arrest can occur even if sinus rhythm persists; an example of 'power failure' without 'electrical failure'.<sup>2</sup> I know of several occasions where failure immediately to distinguish between the two had tragic consequences. Indeed, a pulse oximeter and an automatic sphygmomanometer alone and without an ECG are today adequate for most general anaesthesia. Secondly, there should also be an audible signal with each arterial pulse during invasive blood pressure monitoring with an *easily* adjustable systolic pressure alarm; the diastolic pressure is, of course, clinically unimportant in this context. This is essential even for cardiac surgery when the heart is not visible, since the anaesthetist (or intensive care nurse) cannot watch the monitor continuously or without essential interruptions.

Reliable pulse oximetry in vasoconstricted or low cardiac output patients is another major need. Can we have a sensor for such patients which works from a deep mucosal surface such as the oesophagus, or even maybe from well inside the auditory meatus with a suitable earpiece? As some workers have pointed out, the Ohmeda pulse oximeter works well on the buccal mucosa.<sup>3</sup> Finally, I hope manufacturers will continue to improve the artefact filters on their alarms, a real headache!

20 Hocroft Avenue,  
London NW2

A. GILSTON

## References

1. GILSTON A, RESNEKOV, L. *Cardio-respiratory resuscitation* London: Heinemann Medical, 1971.
2. HELLERSTEIN HK, TURRELL DJ. The mode of death in coronary artery disease. An electrocardiographic and clinicopathologic correlation. In: Surawicz B, Pellegrino ED, eds, *Sudden cardiac death*. New York: Grune and Stratton, 1964, Ch. 3.
3. BARDOGCZY GI, ENGELMAN E, D'HOLLANDER AA. One more use of a vinyl glove: ready-made protection for the reusable oximeter monitoring probe. *Anaesthesiology* 1989; 71: 999.

## Midazolam withdrawal syndrome

The paper by Drs Mets, Horsell and Linton on midazolam-induced benzodiazepine withdrawal syndrome (*Anaesthesia* 1991; 46: 28-9) provides a further indication for the use of isoflurane sedation in the intensive care unit.<sup>1,2</sup> Although our primary use of isoflurane has been in sedating postcardiac surgical patients for relatively short periods of time, a further indication is its use for short-term sedation during withdrawal of a longer term intravenous infusion of a sedative agent. The isoflurane often provides a period of sedation during which the intravenous agent is metabo-

lised/excreted and is therefore a useful buffer against the withdrawal syndrome.

Grobby Road Hospital,  
Leicester LE3 9QE

I. McLELLAN  
E. DOUGLAS

## References

1. BEECHY APG, HULL JM, McLELLAN I, ATHERLEY DW. Sedation with isoflurane. *Anaesthesia* 1988; 43: 419.
2. McLELLAN, I. Isoflurane compared with midazolam in the intensive care unit. *British Medical Journal* 1989; 299: 259.

## Failure of an Ohmeda Oxicap 4700

We read with interest the report of a failure of the Ohmeda Oxicap 4700 (*Anaesthesia* 1990; 45: 1093). We have experienced the same failure and can confirm that the problem occurs when the monitor is switched on after the probe has been placed on the finger. We contacted the manufacturers who had received several reports of this fault from users of the monitor in other countries. They had circulated a warning letter to all users of the monitor which unfortunately we had not received.

The failure of this monitor and a similar failure of a

Kontron 7840 reported previously<sup>1</sup> illustrate the need to observe the patient at all times.

Department of Anaesthesia,  
University of Sheffield Medical School,  
Sheffield S10 2RX

S.P.W. SPIERS  
R.W. HARRIS

## Reference

1. BROOME IJ. A dangerous failure of a pulse oximeter. *Anaesthesia* 1990; 45: 166.

## 'Finger-painting' and the pulse oximeter

Clayton and colleagues (*Anaesthesia* 1991; 46: 3-10) have examined the performance of pulse oximeters under conditions of poor perfusion and found considerable variability in measurements made on patients who had undergone cardiopulmonary bypass. I have recently experienced difficulty in obtaining accurate measurement of oxygen saturation (SpO<sub>2</sub>) for an altogether different reason. A 3-year-old

Asian child presented for a minor surgical procedure which was to be performed as an emergency. After induction of anaesthesia, a pulse oximeter probe (Datex) was placed on the index finger of one hand. After stabilisation, a satisfactory oximeter waveform was obtained with a heart rate identical to that registered by the ECG monitor. SpO<sub>2</sub> was recorded as 63%. A rapid check revealed that the patient's

# Baillière's International Practice and Research CLINICAL ANAESTHESIOLOGY

## Editorial Board

A.R. Aitkenhead (U.K.), M.J. Cousins (Australia), J.F. Crul (The Netherlands), J.G. Jones (U.K.),  
J.F. Nunn (U.K.), L. Strunin (Canada), D. Thomson (Switzerland), H. Van Aken (Belgium)

*Baillière's Clinical Anaesthesiology* is the essential reference for the busy clinician. Each issue is devoted to a separate topic and provides comprehensive coverage from experts in the field. Containing up-to-the-moment reviews, this international reference provides an invaluable source of current clinical opinion, procedures and techniques in anaesthesiology, supported by relevant research data.

## THE 1991 ISSUES

### No. 1 INFECTION AND THE ANAESTHETIST

Guest Editors:  
C.P. Stoutenbeck (Holland)  
& H.K.F. Van Saene (UK)

### No. 2 INTRAVENOUS ANAESTHESIOLOGY - WHAT IS NEW?

Guest Editors: J.W. Dundee (N.Ireland)  
& J.W. Sear (UK)

### No. 3 KINETICS OF ANAESTHETIC DRUGS IN CLINICAL ANAESTHESIOLOGY

Guest Editor: P.F. White (USA)

## PREVIOUS ISSUE TITLES TO COMPLETE YOUR REFERENCE

### Volume 4, 1990

No. 1  
AUTOMATED ANAESTHETIC RECORDS  
Guest Editor: G. Kenny (U.K.)

No. 2  
INTENSIVE CARE:  
DEVELOPMENTS AND CONTROVERSIES  
Guest Editor: G. Dobb (Australia)

No. 3  
ANAESTHESIA FOR DAY CARE SURGERY  
Guest Editor: T.E.J. Healy (U.K.)

### Volume 3, 1989

No. 1  
ANAESTHESIA FOR THE COMPROMISED HEART  
Guest Editor: P. Foex (U.K.)

No. 2  
METABOLIC RESPONSE TO SURGERY  
Guest Editor: D.R. Bevan (Canada)

No. 3  
DEPTH OF ANAESTHESIA  
Guest Editor: J.G. Jones (U.K.)

## ACKNOWLEDGED BY THE MEDICAL PRESS...

"A plethora of clinically applicable facts and therapy" ... "An excellent source of reference for all clinicians" ... "Up to date" ... "Well written" ... "Practical" ... "Well produced and illustrated" ... "Readable" ... "Well balanced" ... "Excellent source of reference" ... "Concise" ... "Comprehensive references" ... "Excellent value" ... "Cost effective" ...

Publication: Volume 5 1991 (3 issues)

Subscription Rate: £41.00 (U.K. and Eire)/£47.00 (All other areas). Single Issues: £27.50 (All areas)



Published by Baillière Tindall  
Harcourt Brace Jovanovich, Publishers



## ORDER FORM: Baillière's Clinical Anaesthesiology

Please return to: Journals Marketing Dept., Harcourt Brace Jovanovich Ltd.,  
24-28 Oval Road, London NW1 7DX, U.K. Tel: 071-267-4466

ISSN: 0950-3501

Publication: Volume 5, 1991 (3 issues)

Prepayment is required

☐ Please enter my subscription for Volume 5, 1991

Back volumes and issues are available  
at current year prices.

☐ Please send me \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

### PRICES:

Annual Subscription: £41.00 (U.K. and Eire)/£47.00 (All other areas).

Single Issues: £27.50 (All areas)

☐ I enclose a cheque for £.....  
(made payable to Harcourt Brace Jovanovich Ltd.)

☐ I wish to pay by credit card Amount £.....  
Diners Club / American Express / Visa (Barclaycard) /  
MasterCard (please delete as appropriate)

Card No. \_\_\_\_\_

MasterCard users should add the numbers appearing above their name

Expiry Date \_\_\_\_\_

Signature \_\_\_\_\_

(Your signature is essential, especially when ordering by credit card)

Name \_\_\_\_\_

Address \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_ Date \_\_\_\_\_ JMD/0291/A781



IMD/0390/A788

lips and tongue appeared pink and no abnormality of gas delivery was detected. The oximeter probe was tried on other fingers with similar results. At this point the patient's hand was turned over and the palms and fingers were found to be covered in a mixture of paints, predominantly blue. No paint was visible on the dorsum of the hands. Subsequently, satisfactory oximetry readings ( $\text{SpO}_2$  97–99%) were obtained from the toes and by placing the oximeter probe 'sideways' across the finger so that the light path by-passed the finger pulp. Postoperative enquiry

revealed that the patient (who had not been premedicated) had been 'finger-painting' in the ward shortly before his operation!

Dyes injected into the circulation (methylene blue and indocyanine green) can cause falsely low values of  $\text{SpO}_2$  and nail varnish may reduce signal amplitude. Perhaps we need to add poster paints to this list.

Booth Hall Children's Hospital,  
Manchester M9 2AA

J.R. SNEYD

### Research difficulties for junior anaesthetists

A recent editorial (*Anaesthesia* 1990; 45: 909–10) reminded me of difficulties that I had to face as a registrar in a teaching hospital trying to combine research with basic specialist training and preparing for the Fellowship examination. Juniors in district general hospitals perhaps face more difficulties. Research has become more and more the means to improve curriculum vitae and augments promotion prospects.

As the author suggests, 'the project must be well supervised and there must be sufficient time and resources to ensure it is completed'. When I commenced my project, senior colleagues were very helpful. I wanted to investigate the clinical impression that a mixture of bupivacaine and fentanyl or diamorphine administered epidurally provided better analgesia for sacral pain during labour than bupivacaine alone. I was a novice in the field of research, therefore discussed the idea with senior colleagues. A protocol for the study was formulated and the hospital ethics committee approved. As the research progressed it became clear that the design of the study was not without flaws. Every patient receiving epidural analgesia during the first stage of labour was a potential entrant into the study. If labour lasted less than 3 hours or sacral pain was not experienced, the patients were retrospectively excluded from the study. However, only a small proportion of patients developed sacral pain and the stringent protocol made recruitment

very slow! The nature of the study demanded my continuous presence in the labour ward, which was not always possible because of service commitments. I had to rotate to specialist units in the city to complete my basic specialist training, during which the project could not be continued. It is correctly pointed out by the author that 'rotational training schemes are not conducive to research'. I struggled and at the end of one year was able to collect some inconclusive data since only 21 patients completed the study. There was a trend which might be interpreted as showing better analgesia in opioid groups, but statistically these differences were not significant.

To further their career, many juniors have to move to other cities, which makes completion of research projects impossible. I was the victim of similar circumstances and am now in Leeds, working as a clinical fellow in cardiothoracic anaesthesia and do not have any exposure to obstetric anaesthesia. In spite of enthusiasm, investment of time, senior guidance and ethics approval, it may be impossible for junior anaesthetists to complete a research project.

I would quote the author again and ask the question, 'Where does this leave the junior researcher?'

General Infirmary at Leeds,  
Leeds LS1 3EX

T.M. AKHTAR

### 'Hazards' of oxygen therapy during spinal anaesthesia

The use of supplemental oxygen therapy during major conduction anaesthesia is a common practice, especially when sedative drugs are given. However, like any therapy, clinicians must be ever alert to potential hazards that may accompany it. We report two unrelated incidents which highlight these potentials.

*Case 1.* An 80-year-old man was scheduled for transurethral resection of the prostate (TURP). No premedication was given. A lumbar subarachnoid block was performed using 3.5 ml plain bupivacaine 0.5%. Oxygen was administered via a plastic facemask at 6 litres/minute and surgery proceeded uneventfully. No sedation was given. Approximately 20 minutes later, an anaesthetic technician noted that the isoflurane vaporizer needed replenishing and asked that he could exchange it for a full one. He placed a full vaporizer on the anaesthetic machine without realising that it had been left on inadvertently. Some time later, the patient began to snore loudly and the patient's jaw was supported. A discussion of the soporific effects of sensory de-afferentiation followed. To relieve fatigue, a technique of flexing the anaesthetist's elbows and leaning on the patient's pillow (resulting in close proximity of the anaesthetist's and patient's faces) was demonstrated. Only then was the odour of isoflurane detected. The patient had been breathing 4% isoflurane via a loose-fitting facemask, which resulted in general anaesthesia.

*Case 2.* A 68-year-old man was scheduled for TURP. Temazepam 20 mg and metoclopramide were given for premedication. A lumbar subarachnoid block was performed using plain bupivacaine. A new plastic facemask was removed from its wrapper and oxygen was administered at 6 litres/minute. Less than 10 minutes later, the patient began coughing violently and a small piece of soft plastic appeared at the mouth. The patient then said that he felt he was 'choking'. The plastic was examined and found to be a piece of the plastic wrapping from the oxygen mask. The fragment was left behind when the wrapping was vigorously torn off the mask.

Both patients made uneventful recoveries. These incidents serve to remind us that '... vigilance is as important during regional anaesthesia as during general anaesthesia'.<sup>1</sup>

Royal Perth Hospital,  
Perth,  
Western Australia 6000

R.H. RILEY  
K.A. HAMMOND  
M.S. CURRIE

### Reference

1. BROWN DL, CARPENTER RL, MOORE DC, BRIDENBAUGH LD, RUPP SM, RAMSEY DH, THOMPSON GE, MULROY MF. Cardiac arrest during spinal anaesthesia. III. *Anesthesiology* 1988; 68: 971–2.

### Angina haemorrhagica bullosa causing respiratory obstruction postoperatively

We wish to report an unusual cause of respiratory obstruction, namely, a large blood-filled blister arising from the soft palate diagnosed to be angina haemorrhagica bullosa. A 78-year-old man was admitted for elective removal of a lipoma in the left supraclavicular region. Before his admission he had been well. A full blood count, liver function tests, urea and electrolytes, chest X ray and electrocardiogram were all normal. He was premedicated with 20 mg papaveretum and 0.4 mg hyoscine intramuscularly. Anaesthesia was induced with thiopentone 200 mg followed by suxamethonium 100 mg. A size 9.0 Portex cuffed oral tracheal tube was passed atraumatically. The patient was allowed to breathe 30% oxygen in nitrous oxide and enflurane.

Following uneventful removal of the lipoma his trachea was extubated when almost fully awake. His breathing was not obstructed at this time. After a few minutes in the recovery area the patient complained of a lump in his mouth. Examination revealed a grape-size blood-filled blister over the junction of the soft and hard palate.

This swelling expanded over the next 15 minutes until it was 4–5 cm in diameter. The patient was very distressed by the swelling and we were concerned as to the possible risk of respiratory obstruction, although his breathing was not at this time impaired. The blister then ruptured spontaneously and discharged a quantity of blood. No further bleeding occurred.

On examination he was noted to have other oral blood-filled blisters. Further questioning revealed that he had had similar lesions in the past which he attributed to cheek biting. A clinical diagnosis of haemorrhagica angina bullosa was made.

This term was first used by Bedham in 1971.<sup>1</sup> The lesions were described as blood-filled blisters arising on the soft palate, the epiglottis, the arytenoids, the pharyngeal wall and the oesophagus. Hopkins and Walker<sup>2</sup> described a series of patients with similar lesions; these mostly ruptured spontaneously with the discharge of blood into the mouth.

They were otherwise well with no serious sequelae reported.

These blood blisters must be distinguished from other bullous lesions of the mouth such as dermatitis herpetiformis, bullous lichen planus,<sup>3</sup> pemphigus<sup>4</sup> and pemphigoid. These diseases tend to have systemic effects and give rise to chronic problems. Our patient did not show these features.

Respiratory obstruction is the most important implication of angina haemorrhagica bullosa. Insertion and removal of a tracheal tube was sufficient trauma to cause this blister. Lesions located in the posterior pharynx close to the larynx, the epiglottis or fauces might produce significant airway obstruction or even asphyxia. Once the diagnosis has been made by exclusion of the other causes, the blister may be ruptured without fear of further dangerous bleeding.<sup>2</sup> Chlorhexidine mouth washes can be used to prevent secondary bacterial infection.<sup>2</sup>

A history of oral blood blisters should alert the anaesthetist to this problem so that extra care may be taken during intubation or the insertion of oral airways. In the recovery room a careful watch should be kept on susceptible patients.

St George's Hospital,  
London SW17 0QT

S.I. HOSAIN  
G. BOUNDS  
J. STANFORD

### References

1. BEDHAM NJ. Blood blisters and the oesophageal cast. *Journal of Laryngology* 1967; **81**: 791–803.
2. HOPKINS R, WALKER DM. Oral blood blisters—angina haemorrhagica bullosa. *British Journal of Oral and Maxillofacial Surgery* 1985; **23**: 9–16.
3. COOKER BED. The diagnosis of bullous lesions affecting the oral mucosa. *British Dental Journal* 1960; **109**: 83–138.
4. SNEDDON IB, CHURCH MD. Diagnosis and treatment of pemphigoid: report on 22 cases. *British Medical Journal* 1955; **II**: 1360–4.

### Modification of laryngeal mask airway

The laryngeal mask airway (LMA) is a new concept in the management of the upper airway. Our experience with the use of the LMA showed one technical problem which is consistent with all sizes. The tube protrudes too far from the mouth of most patients. We cannot see any advantage of this extra length, but it does have some disadvantages. Firstly, accidental dislodgement can occur, especially in the younger child where there is a substantial portion of the tube protruding from the mouth.<sup>1</sup> Secondly, the tube can be kinked especially if monitoring devices are attached close to the patient. Thirdly, it extends close to the surgical field during operations on the head and neck or upper chest. Finally, it can only be fixed to the chin and not to the forehead. We wish to describe our early experience using a modification of this mask which aims at changes in the tube to solve the above problems.

One hundred and fifty adult patients who were not edentulous were included in the study. Anaesthesia was induced with either intravenous thiopentone or propofol and maintained using an inhalational agent in nitrous oxide and oxygen. The size of the LMA was chosen according to the manufacturer's recommendation. When anaesthesia was judged to be adequate the lubricated LMA was inserted using the recommended technique<sup>2</sup> and then the cuff inflated. It was noted that the mask popped out slightly on

inflation of the cuff. Successful placement was indicated by a gentle squeeze of the reservoir bag which resulted in air entry to both lungs with no entry into the stomach and either no or minimal air leak heard around the mask. If a major air leak or failure to ventilate the lungs was detected, the LMA was removed and another trial of insertion was carried out. If a major air leak persisted the patient was excluded from the study. The tube was marked opposite to the upper and lower teeth immediately after insertion. The LMA was removed after recovery and the distance from the upper margin of the cuff to the upper and lower teeth marks were measured. LMAs size 3 were inserted in 70 patients with a male to female ratio of 1:6, a mean age of 49.3 years (SD 14.5) and a mean weight of 56.4 kg (SD 3.5). The mean distance to the upper teeth was 10.5 cm (SD 1.0) and to the lower teeth 9.1 cm (SD 0.9). LMAs size 4 were inserted in 80 patients, with a male to female ratio of 17:3, a mean age of 46.9 years (SD 14.6), a mean weight of 81.8 kg (SD 8.7). The distance to the upper teeth was 11.6 cm (SD 0.9) and to the lower teeth was 10.1 cm (SD 0.9).

Our modification involves cutting the silicone tubes of sizes 3 and 4 LMAs to the mean length of the upper teeth for each size minus two standard deviations, i.e. 8.5 cm for size 3 and 9.8 cm for size 4. A non-kinkable corrugated tube

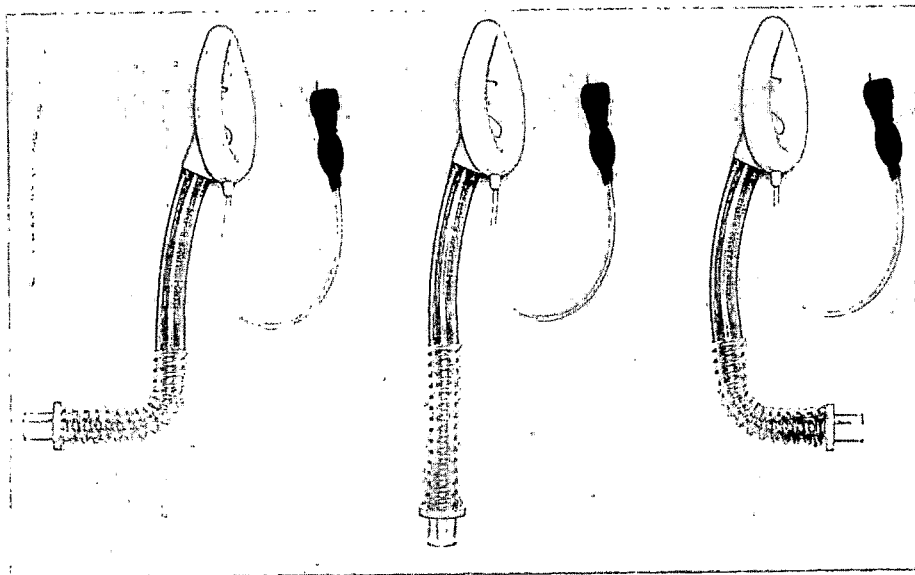


Fig. 1.

8 cm in length and with a 15 mm internal diameter produced by Siemens-Elma company was connected and sealed to the tube of the LMA through a 4-cm male-male connector prepared in our medical engineering department. This design of tube allows bending the part of the tube outside the mouth to any direction without obstruction (Fig. 1).

We have had the opportunity to assess this prototype on 30 patients using both sizes and it has proved to be satisfactory. Insertion and correct placement of this type of LMA is easy to achieve. It can still be inserted by a blind technique. The shaft has to be grasped at the junction of the two tubes and pushed into the mouth with the usual recommended insertion technique. After insertion it is possible to strap the tube to the centre of the chin, the cheek, the forehead or to either side of the mouth, without

kinking. This eliminates the slight possibility of dislodgement. The connector between the two tubes gives strength to the tube and acts as a bite-block as it is opposite to the teeth for most patients. Therefore an additional airway or bite-block is not required. The new tube and the connector are autoclavable.

We are conducting a larger study to include LMAs size 1 and 2 and the edentulous patients.

Southampton General Hospital,  
Southampton SO0 4XY

M.Z. MICHEL  
M.M. CROSSE

#### References

1. MANSON DG, BINGHAM RM. The laryngeal mask airway in children. *Anaesthesia* 1990; **45**: 760-3.
2. BRAIN AIJ. The laryngeal mask—a new concept in airway management. *British Journal of Anaesthesia* 1983; **55**: 801-5.

#### Problems with the 32-gauge spinal catheter

We read with interest the evaluation of 32-gauge spinal catheters through 26-gauge needles by Drs Nagle, McQuay and Glynn (*Anaesthesia* 1990; **45**: 1052-4). We have also encountered several problems with the passage of this size catheter, particularly kinking and flattening of the catheter, which occluded the lumen, and complete breakage of the catheter close to the syringe end.

The 32-gauge catheter is marked in centimetre increments, but the needle supplied in the Microspinal pack is not. This leads to difficulty in determining needle tip-to-skin distance and therefore affects the accuracy of placing less than 2 cm of the catheter in the subarachnoid space. Marking the needle in 1 cm increments, as with the Tuohy extradural needle, would aid placement of the correct length of catheter in the space and would also help when

feeding the catheter during needle removal. We also encountered kinking of the catheter at the hub end of the needle during retraction of the needle over the catheter. Luckily, we were able to thread the needle over the kink in the catheter, and injection through it was not impeded.

However, we later experienced a catheter problem which did result in inability to inject through it. In this case the catheter became flattened in a short segment, by being pinched between the syringe and the bed during transfer of the patient to the recovery room. This weakened the catheter, leading to breakage when attempting to straighten out the flattened segment.

Altnagelvin Area Hospital,  
Londonderry BT47 1SB

E.P. MCCOY  
G. FURNESS

#### Safe use of propofol in a child with acute intermittent porphyria

I should like to present a report of the successful use of propofol for general anaesthesia in a child with acute intermittent porphyria.

A 7-year-old girl, known to have intermittent porphyria, presented for tonsillectomy. She weighed 22.5 kg, was fit

and was not taking any medication.

No premedication was given other than EMLA cream applied to the dorsum of the hand. In the anaesthetic room, routine monitoring of ECG, arterial blood pressure and pulse oximetry was started. A 22-gauge cannula was

inserted and anaesthesia induced with propofol 45 mg. Suxamethonium 25 mg was then given and the trachea intubated with a 6.5 RAE tube. Anaesthesia was maintained with the patient breathing spontaneously a mixture of 33% oxygen and 67% nitrous oxide and boluses of propofol to a total of 150 mg. Blood pressure, heart rate and oxygen saturation remained stable throughout the procedure, which lasted 10 minutes. The patient was awake

within 5 minutes of the end of surgery. There were no sequelae and she was discharged the following day.

I believe that this case adds support to the growing weight of evidence that propofol is a safe agent to use in acute intermittent porphyria.

St James University Hospital,  
Leeds LS9 7TF

A.S. CHRISTIAN

#### Explanation for the overestimation of oxygen saturation seen using nasal pulse oximetry

We were interested to read Drs Rosenberg and Pedersen's paper (*Anaesthesia* 1990; 45: 1070-1) in which they showed that nasal pulse oximetry overestimates oxygen saturation. We would like to suggest another possible explanation for their findings.

A pulse oximeter estimates arterial oxygen saturation by electronically resolving the output from each of its photodiodes, detected by the photodetector, into both alternating current (which detects absorption due to the pulsating component of the tissues, e.g. arterial blood vessels) and direct current (which detects absorption due to non-arterial blood and tissue).<sup>1</sup> The direct current component of the signal should be totally excluded from the further processing which produces values which represent arterial oxygen saturation. However, it has been shown that this is not the case, the presence of nail polish<sup>2</sup> or skin dyes<sup>3</sup> (both non-pulsatile causes of absorption) can seriously affect the accuracy of finger pulse oximetry. There is no reason to assume that the venous and capillary blood within the tissues does not act in the same manner. In areas of low

blood flow (i.e. the finger) the nonarterial blood may be more desaturated than in areas of high flow (i.e. the nose).

The absorption of light by capillary or venous blood with its subsequent effect on the calculation of oxygen saturation could therefore account for the results seen.

Department of Anaesthetics,  
University of Sheffield Medical School,  
Sheffield S10 2RX

I.J. BROOME  
C.S. REILLY

#### References

1. TAYLOR MB, WHITWAM JG. The current status of pulse oximetry: clinical value of continuous noninvasive oxygen saturation monitoring. *Anaesthesia* 1986; 41: 943-9.
2. COTE CJ, GOLDSTEIN EA, FUCHSMAN WH, HOAGLIN DC. The effect of nail polish on pulse oximetry. *Anesthesia and Analgesia* 1988; 67: 683-6.
3. GOUCKE R. Hazards of henna. *Anesthesia and Analgesia* 1989; 69: 416-7.

#### Modification of the Down's adjustable flow generator

One problem with the Down's adjustable flow generator is that the minimum attainable  $F_{IO_2}$  is usually above 0.3 (*Anaesthesia* 1988; 43: 766-9). This is still sometimes too high for patients dependent on the hypoxic drive to stimulate respiration, but only a simple modification is required to produce oxygen concentrations of 24 to 28%. The oxygen-specific probe can be unscrewed and replaced with a 400 kPa air probe enabling the generator to be powered from the pipeline supply of medical air. A short length of 22-mm disposable breathing hose can be used as an inlet manifold on the entrainment port, and oxygen ducted into it via green bubble tubing. In theory, at a total flow of 100 litres/minute, each additional litre of oxygen will raise the concentration by about 1%. In practice, the entrainment

ratios were affected by the value of the PEEP valve used and by the resistance of the arrangement on the entrainment port. However, over the range of PEEP valves from 0.25 to 1 kPa, the flow from a 0-15 litres/minute Rotameter was sufficient to produce oxygen concentrations of between 21 and 35% with the  $F_{IO_2}$  valve set to maximise entrainment.

This modification has been used without problems and appears to be a safe and inexpensive method of extending the use of the generator to wean chronic bronchitic patients from artificial ventilation.

Walsgrave Hospital,  
Coventry

G.C. FISHER

#### Oesophageal rupture and cricoid pressure

About 10 years ago I enquired in *Anaesthesia* whether oesophageal rupture during cricoid pressure had ever been reported,<sup>1</sup> and now Drs Ralph and Wareham (*Anaesthesia* 1991; 46: 40-1) have become the first to describe such a case. This complication would therefore seem to be extremely rare. However, based on this one case, they make the statement, 'Cricoid pressure should be released immediately if active vomiting occurs'. Are they justified in saying this? I have found that most anaesthetists and ODAs are trained to maintain cricoid pressure actively during induction, even if vomiting starts to occur. The concept of 'light application' is not practical unless one has a special meter.

Death as a result of asphyxia or aspiration of vomit still occurs and the Confidential Enquiries into Maternal Deaths regularly report deaths due to inadequate cricoid pressure. Maybe CEPOD will also detect such cases (as well as cases of oesophageal rupture). However, there is no other data on the overall effectiveness of cricoid pressure. I wonder how many lives have been saved by continuing cricoid pressure when vomiting starts just before full muscle relaxation has occurred? If the principle of releasing cricoid pressure on active vomiting is established, I wonder how many patients will die as a result of the pharynx flooding as opposed to dying from oesophageal rupture due to con-

tinued cricoid pressure?

Anaesthesia is a balance of risks. Asphyxia and aspiration are common compared to oesophageal rupture. I urge that cricoid pressure should be maintained if vomiting starts to occur, until the patient is in a lateral, head down position, or intubated.

James Paget Hospital,  
Great Yarmouth NR31 7EB

W. NOTCUTT

#### References

1. NOTCUTT WG. Rupture of the oesophagus following cricoid pressure. *Anaesthesia* 1981; **36**: 911.

#### Bleeding postbronchoscopy

Severe bleeding is well recognised as a complication of the biopsy of suspicious lesions at rigid bronchoscopy. However, trainee anaesthetists may not appreciate that the bleeding is not necessarily revealed as a profuse haemoptysis.

A 73-year-old man suffering from worsening dyspnoea was admitted for rigid bronchoscopy and biopsy of a right-sided hilar mass visible on chest X ray. Following pre-oxygenation and atropine 0.3 mg anaesthesia continued with increments of propofol and suxamethonium to a total of 130 mg and 100 mg, respectively. The lungs were inflated by means of a Sander's injector. The procedure and initial recovery appeared uneventful. After approximately 5 minutes the patient became slightly cyanosed and developed an uncoordinated respiratory pattern. Despite administration of 100% oxygen via a Mapleson C circuit, the cyanosis worsened considerably over the next minute and his conscious level deteriorated. Masseter tone would not permit intubation without muscle relaxation and he was therefore given etomidate 10 mg and suxamethonium

75 mg to facilitate tracheal intubation. Subsequent ventilation of the lungs with 100% oxygen produced poor movement of the left side of the chest and no movement of the right hemithorax. Cardiorespiratory arrest ensued and, fortunately, a cardiac output was restored within a minute by external cardiac massage. A fiberoptic bronchoscope was passed down the tracheal tube and a large blood clot was retrieved which had completely obstructed the right main bronchus. Following cessation of bleeding the patient's trachea was extubated and he recovered uneventfully.

This case demonstrates the need for close observation of patients recovering from a bronchoscopy. Large numbers of rigid bronchoscopies may appear on a thoracic operating list, and notwithstanding the brief surgical time involved, it is unwise to consider them as minor procedures.

11 Priory Farm Close  
Grassendale,  
Liverpool L19 3RS

C.A. HODGSON

#### Acute choreoathetoid reaction to propofol

A 47-year-old woman presented for minor surgery. She was fit and well with no serious medical conditions and no history of anaesthetic problems. Premedication was with temazepam 10 mg one hour pre-operatively and she was quite relaxed on arrival in the anaesthetic room. ECG and noninvasive blood pressure monitoring were instituted and anaesthesia was induced with an intravenous injection of propofol. The injection was administered in accordance with the manufacturer's instructions at a rate of 4 ml every 10 seconds until loss of verbal contact with the patient. Immediately after losing verbal contact the patient began to move involuntarily on the operating table and a further increment of 2 ml was given. The patient moved all four limbs in a violent writhing motion which necessitated her being restrained by the nurses present. The incident subsided spontaneously after a period of about 3 minutes. There were no cardiovascular changes during this time and

there was no indication that she was aware or in pain. Her pupils remained equal, central and small in size, and her respirations were spontaneous and unlaboured. The anaesthetic was continued using oxygen, nitrous oxide and isoflurane, and proceeded uneventfully. Postoperatively in the recovery ward the patient denied any recollection of events or any discomfort on induction.

This case was reported to the Hospital Adverse Reaction Service and the Committee on Safety of Medicine, who have a small number of similar cases on record. This case is reported as the involuntary movements were of a severe and sustained nature and could possibly have led to some physical damage to the patient if she was not restrained.

Broadgreen Hospital,  
Liverpool L14 3LB

P. McHUGH

#### Misdiagnosis caused by an epidural dressing

A 17-year-old man was admitted with crush injuries to his chest and abdomen caused by a crane hook. On arrival he was found to have fractures of two ribs on the left side, with underlying pulmonary contusion, and intra-abdominal haemorrhage. At laparotomy, a bleeding artery in the mesentery was found and ligated and a large bowel resection was performed for a colonic perforation. The total blood loss was estimated to be approximately 4 litres. The routine swab count at the end of the operation revealed that an 18 inch by 18 inch abdominal pack was missing. This could not be found despite a careful search of the

abdomen. A check X ray on the table failed to demonstrate the retained pack, although the radiograph did not give a complete picture of the upper abdomen.

Postoperatively, the patient was admitted to the intensive care unit for controlled ventilation and a thoracic epidural was inserted at the T<sub>9-10</sub> interspace for analgesia. His trachea was extubated later the same day and he was transferred to the high dependency unit the following morning in good condition. On the first postoperative day, a chest X ray demonstrated the presence of radiopaque lines in the region of the upper abdomen. These were

thought to represent the missing pack. The operating theatre was therefore arranged for re-exploration. Fortunately, further pre-operative examination in the anaesthetic room revealed that the radiographic appearance was caused by the radiopaque marker of a green Raytec swab used to dress the epidural site. Removal of this

followed by a repeat X ray obviated the need for laparotomy. This potentially dangerous scenario demonstrates the inadvisability of using radiopaque swabs as dressings.

Wythenshawe Hospital,  
Manchester M23 9LT

A.M. BERRY  
M.R. PATRICK

### The Woolley and Roe case

In 1983 I visited Chesterfield and recorded a conversation with Dr J.M. Graham, the anaesthetist who administered the spinal anaesthetics to Woolley and Roe<sup>1,2</sup> in 1947. We agreed that the damage to the patients was not similar to that produced by phenol and that 'invisible cracks' theory of leakage of phenol into three consecutive ampoules on the same day was too unlikely to be credible. Dr Graham recalled that, 'We did all sorts of experiments banging these things (20 ml ampoules of light nupercaine) about and we could not crack one. We could smash them, but we could not crack them. They (Nuffield Department of Anaesthetics, Oxford) produced some (ampoules with invisible cracks) in court for me to look at, and said "Would you see that crack?" You could see it if you looked jolly carefully, you could eventually find it ... and I said, "These are thermal cracks, are they not?" (made by touching the ampoule with a hot wire) ... They had to admit that you could not crack them. They either broke altogether or nothing happened.'

Dr Graham was certain that the correct drug from the correct ampoule was administered in each case. Therefore some external factor unrelated to the ampoules was to blame and Dr Graham came very close to the truth. He mentioned that the operating theatre sister was not well at the time of the tragedies and that, on the day in question, she went off duty at lunch time. I could not see any relevance because at least one of the patients, Woolley, was operated on after lunch.<sup>2</sup> 'But, you see,' Dr Graham continued, 'it just depends who you think is the cause of it. Now, if three things happen in one day, there is a common cause, something has gone wrong ... Nobody will believe me, but I still think that it was some sort of infection ... something was contaminated in some way ... how else can you account for it?' When I commented that one would not expect infection to get into three different ampoules on the same day, but never at any other time, Dr Graham replied, 'Ah, no, I was thinking even from coming from outside, if the needles were not sterile, if the syringe was not sterile ... which was the sister's responsibility. I mean, it's only a

suggestion because I can't think of anything else. I am sure that it is not phenol and I am certain it is not using the wrong stuff.' The idea of contaminated needles and syringes was very close to Dr Hutter's explanation; Dr Graham thought of inadequate sterilisation, whereas Dr Hutter suggests sterilisation in a toxic chemical in the steriliser.

Neither Woolley nor Roe received any financial compensation because negligence was not proven.<sup>2</sup> Both men eventually died. Dr Graham said that some years after the trial Mr Roe was admitted to hospital. He was dying and asked to see Dr Graham, who went with some trepidation to the patient's bedside. The following conversation ensued: Dr Graham: 'Good morning, Mr Roe, and how are you?' Mr Roe: 'Not very well, doctor' Dr Graham: 'Yes, I know that, and I am very sorry.' Mr Roe: 'I wanted to see you because I wanted to tell you how sorry I am for all the trouble that I have caused you. I did not want to bring the case at all, it was the union.' Dr Graham: 'Mr Roe, they were quite right. You came in here for a simple operation and you finished up with your legs paralysed. But I would like you to know that to this day I don't know what went wrong. I can promise you that it was not that someone gave you the wrong thing by mistake. We just don't know.' Mr Roe: 'Thank you very much for telling me, doctor.' Dr Graham's final comment on the case was: 'What a marvellous chap (Mr Roe)—to say "I am sorry for all the trouble I have caused you".'

Foothills Hospital at  
The University of Calgary,  
1403 20 Street NW, Calgary AB,  
Canada T2N 2T9

J.R. MALTBY

### References

1. HUTTER, CDD. The Woolley and Roe case. A reassessment. *Anaesthesia* 1990; 45: 859-64.
2. The WOOLLEY and ROE case. Woolley and Roe vs Ministry of Health and others. *Anaesthesia* 1954; 9: 249-70.

### Ketamine does not always work in treatment of priapism

Dr Hutchinson (*Anaesthesia* 1990; 45: 794) suggests that ketamine is a consistently successful treatment for intra-operative priapism.

We report the case of a 68-year-old man who came to surgery for haemorrhoidectomy and a perurethral resection of the prostate. He was anaesthetised with thiopentone followed by atracurium and his lungs ventilated with 1-2% isoflurane in nitrous oxide:oxygen through a laryngeal mask. He developed an erection at the end of the haemorrhoidectomy, which prevented the planned perurethral resection of the prostate. Ketamine 25 mg was given intravenously without success. The surgeon then injected metamaminol 1 mg into the shaft of the penis. Detumescence occurred and the resection of the prostate performed. The arterial blood pressure and heart rate remained stable and no cardiac arrhythmia was noted. The erection partially

reappeared after 30 minutes but surgery was completed.

An understanding of the physiology of penile erection has led in recent years to new treatment strategies in priapism. Therapeutic efforts are now directed on the basis of the underlying pathophysiology.<sup>1</sup> Intra-operative priapism may have to be treated by intracorporeal injection of alpha adrenoceptor stimulants.<sup>2</sup> It should be stressed that it is necessary to monitor continuously both the blood pressure and the pulse rate as complications of alpha adrenergic injection can occur.<sup>3</sup> The use of intracorporeal metamaminol should be considered if priapism interferes with surgery.

Royal Free Hospital,  
London NW3 2QG

B. APPADU  
I. CALDER



## References

1. BENSON HT, LEVENTHAL JB, OVASSAPIAN A. Ketamine treatment of penile erection in the operating room. *Anaesth Analg*. 1983; **62**: 457-58.
2. BLOCK T, STURM W, ERNST G, STAEHLER G, SCHMIEDT E. Metaraminol in the treatment of various forms of priapism. *Urologe* 1988; **27**: 225-29.
3. O'BRIEN WM, O'CONNOR KP, LYNCH JH. Priapism: current concepts. *Annals of Emergency Medicine* 1989; **18**: 980-83.

## Meningitis following spinal anaesthesia

We were interested to read the case report of 'presumed' meningitis following an obstetric spinal anaesthetic (*Anaesthesia* 1990; **45**: 376-7). However, we would suggest a word of caution before performing lumbar puncture in a patient with signs suggestive of meningitis, such as described in the case report.

An 83-year-old woman underwent a total knee arthroplasty performed under spinal anaesthesia plus a light covering anaesthetic. A 22-gauge needle was used for the spinal puncture and she had an uneventful intra-operative course. Immediately upon waking she complained of severe headache and although unusually early, this was thought to be of dural puncture origin. Postoperative observations were satisfactory for 12 hours, at which time she developed fast atrial fibrillation, a fever of 38°C and appeared increasingly difficult to rouse. At this stage there were no focal neurological features and she was treated with digoxin. Over the next 12 hours she developed marked neck stiffness and photophobia, and became unresponsive to verbal stimuli. Her left side showed an equivocal increase in tone and the right plantar was upgoing. A differential diagnosis of meningitis and cerebrovascular accident was considered.

A CT head scan was performed which revealed an area of low attenuation in the right parietal lobe, consistent with a cerebral infarct. A lumbar puncture was not therefore deemed necessary and no antibiotics were given.

With the experience of this case, we suggest performing first a CT scan to exclude intracerebral pathology, rather than lumbar puncture in a patient displaying signs of meningitis postspinal puncture. A case of subdural haematoma following spinal anaesthesia has been described<sup>1</sup> and there is always the risk of neurological deterioration following lumbar puncture in a patient with raised intracranial pressure. Had one been performed first in our patient, she may not have made such a good recovery from her stroke.

Southlands Hospital,  
Shoreham-by-Sea,  
West Sussex BN4 6TQ

J. VAN-ANDEL  
N.G. LAVIES

## Reference

1. BARKER GL. Spinal subdural haematoma following spinal anaesthesia. *Anaesthesia* 1988; **43**: 664-5.

## Book reviews

- Outpatient anesthesia** 428  
 Edited by P.F. WHITE  
**Baillière's Clinical Anaesthesiology, Vol. 4, No. 2:**  
**Intensive care developments and controversies** 428  
 Edited by G.J. DOBB  
**Spinal cord injuries—anaesthetic and associated care** 429  
 Edited by J.D. ALDERSON AND E.A.M. FROST

- Baillière's clinical anaesthesiology; international practice and research. Anaesthesia for day case surgery** 429  
 Edited by T.E.J. HEALY  
**Controversies in obstetric anaesthesia, Number one** 430  
 Edited by B. MORGAN  
**Gas monitoring and pulse oximetry** 430  
 J.S. GRAVENSTEIN

### Outpatient anesthesia

Edited by P.F. WHITE. Pp. xvi + 520. Churchill Livingstone, 1990. £47.50.

Many American anesthesiologists are principally involved in outpatient work, because of the dominance of financial considerations, and this book aims to be their practice and training 'resource'. The chapter on anaesthetic considerations is a short account of 'basic pharmacologic and physiologic principles', with little specifically related to outpatient anaesthesia, as in the case in the accounts of premedication, monitoring, local or general anaesthesia. The descriptions of outpatient management of the old and the young, postoperative pain and complications are more specific. A good description of methods of assessing recovery from anaesthesia ends with the admission that there is no suitable test for discharge of patients so we must rely on clinical acumen. Indeed, the gist of this 520 page volume is contained in the first and last chapters, an overview and controversies in outpatient anaesthesia.

A secondary aim of the book is to be useful to other 'health care professionals involved in ambulatory surgery'. However, the chapters on economic and regulatory facilities and personnel, nursing and surgical matters are brief, vague or parochial and therefore cannot greatly help the general reader tackle practical problems in day care units.

A chapter on legal considerations by lawyers is clear and concise and indicates trends in litigation. In the role of a manager, an anaesthetist director of a day care unit is charged with developing or enforcing policies, protocols and procedures and thus may be held responsible so that extra medicolegal cover is recommended. The lawyers dictum 'if you haven't written it down you haven't done it' must be applied in the light of a comprehensive justification throughout this book that routine pre-operative tests are ill advised, but the clinical history and examination must be thorough. Two chapters describe the use of sedatives and analgesics with local or no anaesthetic, and introduce a second meaning of the acronym MAC (monitoring anaesthesia care). This technique is associated in America with a high complication rate according to a Federated Ambulatory Surgery Association report. A British reader may bridle at the misspelling of the name of Sir Humphrey Davy or quibble over four different erroneous references to the Belfast surgeon, Mr J.M. Nicoll's, 1909 British Medical Association paper. This surgeon gave an important account of his 7392 day-care operations on children (more than half under 3 years of age) and the use of a house near the hospital as a postoperative centre for infants with their mothers. This concept much predated the recent use of recovery centres for intermediate care, where sophisticated costly hospital services are not needed but home care is

difficult. The illustrations, references and index of this book are useful, as are the examples of record charts and treatment guidelines which could be modified for local needs. The book should, therefore, be in reference libraries for enthusiasts to expand their fund of information and to encourage a search for more relevant facts on this expanding subject.

H.T. DAVENPORT

### Baillière's Clinical Anaesthesiology. Vol. 4, No. 2: Intensive care: developments and controversies.

Edited by G.J. DOBB. Pp. x + 613. Baillière Tindall, 1990. £22.50.

The most recent addition to Baillière's excellent series of Clinical Anaesthesiology is a volume devoted to developments and controversies in intensive care.

In our audit-ridden society it is fitting that the opening chapter should cover scoring systems in the intensive care unit (ICU). This is optimistic about the reader's understanding of statistics, but is nevertheless nicely reviewed by Dr S. Willatts. Pharmacokinetics and pharmacodynamics in the critically ill is an excellent section which looks carefully at drug prescription in the ICU on the basis of blood concentration, half-life, volume of distribution and clearance. Disappointingly the authors conclude that such is the complexity of pharmacokinetics in the critically ill that empirically based prescribing and titration of dose against effect is still the method of choice. Nevertheless, the chapter is full of helpful practical information and is complemented by a subsequent section on newer drugs in the ICU. 'The heart and circulation in sepsis' includes a good account of the pathophysiology of this dangerous condition as well as a review of important mediators and therapeutic implications. Drs W. and S. Kox consider oxygen consumption, hypoxaemia and lactate utilisation at a cellular and biochemical level and illustrate the question of critical oxygen delivery by several clear and informative studies. This is followed by an extremely good section on peri-operative myocardial ischaemia covering pathophysiology, infarction as well as pre- and postoperative care. Chapters on airway management and ventilation review current practices and introduce several newer ones such as noninvasive ventilation and airway pressure release. Controversies on intubation, tracheostomy and humidification are re-evaluated and HME's are given the thumbs down. 'Nosocomial infection' by Dr M. Hemmer enlivens a dull subject and describes the problem and its incidence (15–60%). Selective decontamination of the gut comes up for air but no

decision is taken on whether it is a good thing or not. The status remains quo.

'Artificial kidneys' gives the reader a clear account of modern techniques in renal support. Happily, the daunting mathematical approach gives way to a really excellent practical description which reflects the obviously wide experience of the authors. The account of enteral nutrition is written by Dr G. Dobb, Head of Intensive Care in Perth and guest editor. This is a particularly well researched section, which like the rest of the book, contains an exhaustive list of up-to-date references. Dr Dobb takes care to present his nutritious and easily assimilated information on the basis of published studies, with the result that traditional practices and unproven habits (like starter regimens) can now be critically re-appraised.

Rather unexpectedly the penultimate chapter is on the critically ill child. Clearly, this is a big subject but the authors consider only resuscitation of common major life-threatening events. For those units or emergency rooms unused to dealing with paediatric problems there are valuable lessons here on immediate management, perhaps before transfer. Finally, a Cambridge group discuss multiple organ donation. The subject is looked at from scientific, clinical and ethical perspectives and completes a realistic and well-informed book which will become deservedly popular.

R. ARMSTRONG

#### Spinal cord injuries—anaesthetic and associated care

Edited by J.D. ALDERSON AND E.A.M. FROST. Pp. xiii+258. Butterworth Scientific, 1990. £50.

While expertise in the management of spinal cord injuries has been built up in specialised units, these patients may now present, either in the acute phase after the injury or during the chronic rehabilitation phase, to any doctor in any hospital. This book describes the problems presented by these often young subjects and suggests the appropriate modes of treatment to provide optimal care.

The book comprises 13 chapters and 250 pages and is written by 17 authors from the United Kingdom and the USA with one co-author from Finland. The first chapter provides a clear description of the anatomical, physiological and pathological basis of spinal cord injuries. Scientific and research data are applied to clinical situations and pathological syndromes.

Initial care policies for spinal cord injury patients in the UK and USA are contrasted, especially the emphasis on the use of extraction devices and the spinal board in the USA. In the UK a stable lateral position is preferred for the management of unconscious patients with spinal cord injuries to a supine patient on a spinal board. It is reassuring to learn that the incidence of complete neurological lesions has fallen in the USA since the introduction of the well organised, highly trained Emergency Medical services and their personnel.

The chapter on Emergency room care is written by a neurosurgeon who prefers nasotracheal intubation and even cricothyroidotomy to orotracheal intubation. This contrasts with a later chapter by Frost who avoids nasotracheal intubation and quotes a study showing a 45.3% incidence of bleeding and 9.4% incidence of positive blood culture after nasotracheal intubation, whereas only 2.3% has a positive blood culture after the oral route was used. There is no evidence that careful orotracheal intubation increases spinal cord injury. An editorial overview on this subject would have been helpful.

The anaesthetic management of acute spinal cord injuries

summarise the best of current practice and updates the sentinel review articles by Desmond in 1970<sup>1</sup> and Fraser and Edmonds-Seal in 1982.<sup>2</sup> Prophylactic use of nonparticulate antacids and metoclopramide is recommended as described by Fraser and Edmonds-Seal, but now should be supplemented by the administration of H<sub>2</sub> receptor blockers. The chapter on intensive care describes the use of ICU ventilators and ventilatory modes, cardiovascular management, sedation and the avoidance of sepsis. The author strongly recommends sucrofate instead of antacids and H<sub>2</sub> receptor blockers to maintain the gastric acid barrier and yet provide prophylaxis against upper gastrointestinal bleeding. Eighty five per cent of spinal injury patients will develop a state of hyperreflexia especially to transurethral surgery. The use of spinal anaesthesia to control this potentially dangerous problem is advocated.

These main chapters take up about a half of this book. Further chapters cover nursing care, anaesthesia for nontraumatic spinal disease, spinal cord injury patients with pain, trends in research, and moral and legal issues. Although these subjects are interesting, they seem to be provided as ballast in a book that could have been reduced in size. Nevertheless, this is an invaluable and most readable book and will serve as an essential source of reference not only in anaesthetic departments specialising in the management of these patients, but in all good departments.

R. GREENBAUM

#### References

1. DESMOND J. 1970. Paraplegia, problems confronting the anaesthetist. *Canadian Anaesthetists Society Journal*: 17: 435-51.
2. FRASER A, EDMONDS-SEAL J. 1982. Spinal cord injuries. A review of the problems of facing the anaesthetist. *Anaesthesia*: 37: 1084-98.

#### Baillière's Clinical Anaesthesiology; international practice and research. Anaesthesia for day case surgery

Edited by T.E.J. HEALY. Pp. x+823. Baillière Tindall, 1990. £22.50.

This book has been published at the right time. It has 11 chapters, eight written by British contributors and the subject of day case anaesthesia is adequately covered. There is, however, no specific chapter on postoperative analgesia for day patients. This is an important omission since poor pain treatment is a source of inappropriate hospital admission after day surgery.

It is a well produced book and is a suitable text for anaesthetists, surgeons and nurses involved in this expanding field. There are only a few illustrations but the tables are concise and complement the text. The references are both relevant and up-to-date. As with any multi-author book there is considerable overlap. However, this does not detract from excellent chapters on pre-operative assessment, premedication, local anaesthesia and recovery. Since children benefit psychologically from day surgery the commonsense, well-researched review of paediatric day case anaesthesia from Australia is excellent value.

There are three chapters on the organisation of different types of day units. The contribution from Dr Hoskins in New Zealand is a model for others to follow. Oral surgeons have been pioneers in the day surgery field and there is an excellent chapter from Professors Rood and Healey, describing their Manchester Dental Day Unit, with results.

There is a need for cost-effective, quality surgical care in the UK and the recent report from the Audit Commission

on day surgery should expand this field. This textbook is a genuine attempt to educate a multidisciplinary readership. I recommend it to anyone with an interest in day surgery and anaesthesia. Anaesthetic departmental libraries should possess a copy if only to indicate to all readers that there is still a great deal of research to be done on this subject. The guest editor is to be congratulated for assembling such an expert group of contributors, the end result is the production of a worthwhile book.

T.W. OGG

### Controversies in obstetric anaesthesia, Number One

Edited by B. MORGAN. Pp. vi + 138. Edward Arnold, 1990. £16.95.

The cut and thrust of parliamentary debate has become one of our everyday spectacles and compulsive television viewing for some. The popularity of such adversarial presentation has also reached anaesthesia judging by the success of the Queen Charlottes' Hospital meeting on *controversies in obstetric anaesthesia*. This book, based on the formula employed at these meetings, explores a number of controversial areas in obstetric anaesthetic practice by supporting the motion (or perhaps more correctly the statement) with one paper and then presenting a diametrically opposing paper. Whilst such debates are usually exciting to the observer when viewed in the flesh, does such a technique translate well to the written word or is one left with a somewhat lifeless record of two opposing views?

The controversies covered in this book consist of aspects of regional analgesia and anaesthesia, antacid therapy and organisation of practice. The scope of the regional topics can be seen from some of the titles: 'spinal is better than epidural anaesthesia for elective Caesarean section', 'epidural anaesthesia is contra-indicated in mothers on low-dose heparin' and 'midwife top-ups must be abandoned'. The antacid presentations debate the place of magnesium trisilicate in modern practice and the need for all mothers in labour to receive antacid prophylaxis. Perhaps the most contentious sections relate to organisational matters, the two topics being: 'ideally all emergency Caesarean sections should be performed in the delivery room' and 'units in which less than four Caesarean sections a week are done should not be recognised for training of anaesthetists'. It is clear that, with the possible exception of the low-dose aspirin problem which had not raised its head at the time of publication, Dr Morgan has chosen current and important controversies to discuss.

The list of contributors (or combatants) is impressive, including many of the current 'First Division' of obstetric anaesthetists. Each argues their point clearly and succinctly, in well presented papers including sufficient numbers of charts and tables to help the reader without spoiling the flow of the argument. However, with such an adversarial approach in the book, the reader may often find him/herself in the same situation as your reviewer in muttering under one's breath, and occasionally louder, cries of 'rubbish', 'nonsense' or 'exactly' depending upon your alliance. This is not a book that tells you how to do it, but a book that, in the areas covered, makes you question what you do and why you do it. Any book that manages to broaden our minds sufficient to question some of the time-honoured fables of anaesthesia is to be applauded.

However, to return to the original question of whether such an approach works as well in book form as it does in the flesh. The answer is, not quite. What is lacking is the atmosphere of a live debate, the moments of thoughtful

silence, the ripple of astonishment through the audience after a contentious point and the stagecraft of the presenters. However, this should not detract from the value of this book. When two such opposing arguments are used to support a view one suspects that the truth lies somewhere in the middle; however, the editor gives no help in estimating this middle ground and the reader must draw his or her own conclusions and in doing so has fulfilled the intentions of the book—to make us think about the problems.

This book is the first of a series with later volumes covering other controversies in addition to giving short updates on previous topics. Your reviewer eagerly awaits the next volume.

M. HARMER

### Gas monitoring and pulse oximetry

J.S. GRAVENSTEIN. Pp. xi + 148. Butterworth, 1990. £18.

The title and size of this book implies a concise factual account of gas monitoring and pulse oximetry. It is certainly concise. One chapter consists of three pages of which half are diagrams. As far as facts are concerned, they are present but hidden among much unrelated information. Does the title of the book warrant a chart for conversion of Torr to kPa or a table of water vapour pressure at various temperature? The book contains many illustrations, indeed over half of it consists of diagrams. Unfortunately many of the diagrams are repetitive and too detailed. For instance, in the chapter on 'monitoring of the machine' there are six identical half-page diagrams repeated just to demonstrate a leak at various points in a circle system.

The introductory chapter on the concept of monitoring should be read by all medical workers as the general philosophy for gas monitoring is well argued. However, after this thoughtful introduction there follows a disappointingly superficial account of the derivation of various -grams and -graphs. Although Monty Python devotees may appreciate the concept (illustrated) of the capnogram taking the form of an elephant swallowed by a snake, I doubt whether English language purists will favour the introduction of words such as oxy- and anesthetigraphy. Subsequent chapters briefly describe the influence on recordings of gas volumes, solubility, cardiac oscillations and metabolic changes but are they justified in such a titled book? The whole essence of this book evolves around monitoring of gases during regional and general anaesthesia. Indeed, good material is included in these chapters but the text is again littered with spurious information, much of it probably outside the remit of the title. Such potentially good chapters have somehow fallen between two different groups of readers, being of doubtful practical use to the research worker and yet too brief for the inquiring anaesthetist. In a later section the author uses novel diagrams to explain changes in pulse oximeter and capnograph readings following increased mismatch between ventilation and perfusion. However, unless the reader is well acquainted with respiratory physiology, the brief interpretations may be very confusing. It is very disappointing to see in the penultimate chapter that although mass spectrometry, photoacoustic excitation and Raman scattering are described, more common methods of gas monitoring are not mentioned.

In his preface the author states that the book is for the busy clinician. I doubt it. This book is certainly not for quick reference. The author continuously assumes that the reader already has wide knowledge either of anaesthetic practice or of physical measurement. As a result very little

detailed explanation is given and often the legends to the diagrams contains more information than the text. Those anaesthetists not well versed with gas monitoring would be advised to seek respite in more orthodox and detailed tomes, while a research worker setting out to monitor gases would find discussion with an established academic anaesthetist more fruitful. Much of the information in this

book is already available in more detail elsewhere and with the present financial restraints, it might be difficult to persuade a library committee to invest in this book. I still remain unsure as to whom this book is aimed at.

G. HULANDS

## EPIDURAL & SPINAL BLOCKADE IN OBSTETRICS

Edited by

**Felicity Reynolds**, Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.

This book has evolved from the Obstetric Anaesthetist's Association meeting in the UK in September 1989. Unlike other books of proceedings however, the well-known contributors were invited not only to speak but also to provide a chapter reviewing their subject, prepared especially for the book.

Edited by Dr. Reynolds, the result is an interesting blend of a review of the last ten years' developments coupled with presentations of current research and coloured throughout with stimulating, amusing discussion.

- Includes unique illustrations, using state-of-the-art imaging techniques, of the anatomy of the epidural space
- Features excellent reviews of the rapidly expanding fields of Caesarean section and the use of spinal opioids.

The closing section, The Recipient, discusses the effects that conduction blocks may have on the baby, and reviews the lay literature covering consumer's views of epidural practice.

### CONTENTS

The Epidural Space: Anatomy and Approach • Indications and Contraindications to Epidural Blockade • Test Doses • Top-ups Versus Continuous Infusion • Management of the Second Stage of Labour • Local Anaesthetic Toxicity • Taps and Patches • Neurological Complications of Epidural and Spinal Blockade • Regional or General Anaesthesia for Emergency Caesarean Section • Spinal and Epidural Blockade • Chairman's Responses • Epidural Opioids in Labour • Epidural Opioids for Caesarean Section • The Effects of Regional Analgesia • Public Opinions • Summary and Conclusions • Index.

**0 7020 1401 X ca 250pp Ills Pb**  
**August 1990 Baillière Tindall £17.50**

## ESSENTIALS OF ANESTHESIOLOGY 2/e

**David C. Chung**, Consultant, Dept Anaesthesiology, The Mississauga Hospital, Ontario, Canada;

**& Arthur M. Lam**, Assoc. Prof., Dept Anaesthesiology, Harborview Medical Center, Univ. Washington, Seattle, USA.

A primer in anaesthesia written for those about to specialise in the field, this book provides concise, yet thorough coverage of the scientific basis and the principles of safe practice. All chapters have been revised to incorporate new drugs, equipment and medical conditions.

Includes:

- Revised and expanded section on management of postoperative pain
- New section on quality assurance and risk management
- New chapter on AIDS and the anaesthetist
- New section on autologous blood transfusions

**0 7216 3084 7 304pp 50 ills SpPb**  
**1989 W.B. Saunders £15.50**

## TEXTBOOK OF PAEDIATRIC ANAESTHETIC PRACTICE

**E. Sumner & D. Hatch**

**0 7020 1336 6 560pp 103 ills Hb**  
**1989 Baillière Tindall £37.50**

## INTENSIVE CARE: A Concise Textbook

**C.J. Hinds**

**0 7020 1150 9 387pp 109 ills Pb**  
**1986 Baillière Tindall £15.95**

## OBSTETRIC ANAESTHESIA & ANALGESIA 3/e

**D.D. Moir & J. Thorburn**

**0 7020 1154 1 390pp Ills Hb**  
**1986 Baillière Tindall £23.00**

## DIFFICULTIES IN TRACHEAL INTUBATION

**I.P. Latta & M. Rosen**

**0 7020 1096 0 192pp 150 ills Hb**  
**1985 Baillière Tindall £19.95**

**W.B. Saunders & Baillière Tindall**

**HARCOURT BRACE JOVANOVIĆ**

Available from your bookseller. For more information please contact: The Marketing Dept, Harcourt Brace Jovanovich, 24-28 Oval Rd, London NW1 7DX, UK.

OR phone 081 300 3322

Prices and specifications may vary without notice.

# AFFECT THE EYE?



Basal tear production can decline by as much as 95% during general anaesthesia,<sup>1</sup> resulting in drying of the corneal epithelium and painful corneal abrasions.<sup>2</sup>

That's why it's so important to protect the cornea with LACRI-LUBE, the leading ophthalmic ointment for the treatment of dry eye.

LACRI-LUBE supplements deficient tears throughout a surgical procedure, helping to prevent accidental tape adhesion to the cornea or conjunctiva. In long procedures, further applications may be required.



## LACRI-LUBE\*

(preservative free ophthalmic ointment)

FOR CONTINUOUS CORNEAL PROTECTION DURING  
GENERAL ANAESTHESIA

#### LACRI-LUBE\*

**Presentation:** Off-white, smooth, preservative-free sterile ophthalmic ointment containing white petrolatum, mineral oil and non-ionic lanolin derivatives. **Uses:** Useful as adjunctive therapy to lubricate and protect the eye in conditions such as exposure keratitis, decreased corneal sensitivity, recurrent corneal erosions, keratitis sicca, and also in ophthalmic and non-ophthalmic surgery. **Dosage and Administration:** For topical administration. Pull lower lid down to form pocket and apply a small amount as required. There is no variation of dose for age. **Contra-indications, warnings, etc.** No known contra-indications. **Pharmaceutical precautions:** Store away from heat. To avoid contamination during use, do not touch tube tip to any surface. **Legal Category:** P. **Package Quantities:** Lacri-Lube is available in 3.5g and 5g ophthalmic ointment tubes.

\*Trade Mark

And now LACRI-LUBE is available in a new **preservative-free** formulation which eliminates the potential for preservative sensitivities.

Yet another reason for you to choose LACRI-LUBE as an automatic part of your induction routine. After all, who needs eye problems after a hip operation?

**Basic NHS cost:** 3.5g £1.97, 5g £2.49 (as at January 1991). **Further Information:** Dry eye symptoms commonly persist at night - Lacri-Lube has been specifically formulated to lubricate and protect the dry eye during sleep. Lacri-Lube can provide prophylactic ocular care during general surgical procedures as an adjunct to taping of the eyelids. **Product licence number:** 0426/0041

**ALLERGAN PHARMACEUTICALS**

Allergan Ltd., High Wycombe, Bucks HP12 3SH.

**References:** 1. Cross, D.A. et al., *Anesthesia and Analgesia* ... Curr Res 1977; 56 (1): 35-37. 2. Snow, J.C. et al., *Anesthesia and Analgesia* ... Curr Res 1975; 54 (4): 465-467.



# Contents: Anaesthesia, vol. 46, no. 5, May 1991

## EDITORIAL

Ropivacaine

*F. Reynolds*

339

## ORIGINAL ARTICLES

The neuromuscular blocking effect of Org 9426. A new intermediately-acting steroidal nondepolarising muscle relaxant in man

*L.H.D.J. Booij and H.T.A. Knafe*

341

The effect of prophylactic fentanyl on shivering in elective Caesarean section under epidural analgesia

*W.H.D. Liu and M.C. Luxton*

344

Monitoring of irrigating fluid absorption during transurethral prostatectomy. A study in anaesthetised patients using a 1% ethanol tag solution

*J. Hultén, V.J. Sarma, H. Hjertberg and B. Palmquist*

349

Plasma concentrations of bupivacaine after supraclavicular brachial plexus blockade in patients with chronic renal failure

*A.S.C. Rice, C.E. Pither and G.T. Tucker*

354

The Brain laryngeal mask. A comparative study with the nasal mask in paediatric dental outpatient anaesthesia

*R. Bailie, M.B. Barnett and J.F. Fraser*

358

Cardiovascular effects of epidural local anaesthetics. Comparison of 0.75% bupivacaine and 0.75% ropivacaine, both with adrenaline

*H.E.M. Kerckamp and M.J.M. Gielen*

361

Airway protection by the laryngeal mask. A barrier to dye placed in the pharynx

*R.E. John, S. Hill and T.J. Hughes*

366

Topical amethocaine in strabismus surgery

*D.M. Watson*

368

## CASE REPORTS

Caesarean section in undiagnosed Eisenmenger's syndrome. Report of a patient with a fatal outcome

*D.H. Gilman*

371

Hypermetabolism in arthrogryposis multiplex congenita

*P.M. Hopkins, F.R. Ellis and P.J. Halsall*

374

Chickenpox pneumonia, its complications and management. A report of three cases, including the use of extracorporeal membrane oxygenation

*G.P.M. Clark, P.M. Dobson, A. Thickett and N.M. Turner*

376

Paediatric blood pressure and anaesthesia

*C.M.P. Mather*

381

Computer-controlled anaesthesia in the management of bronchopleural fistula

*J.A. Donnelly and R.E. Webster*

383

Traumatic pneumocephalus following head injury. A complication of general anaesthesia

*M.D. Finch and G.A.R. Morgan*

385

## APPARATUS

Pulse oximetry compared with Doppler ultrasound for assessment of collateral blood flow to the hand

*K. Pillow and I.A. Herrick*

388

Single operator cardiopulmonary resuscitation in ambulances. Which ventilation device?

*G.L. Greenslade*

391

A new portable oxygen system using liquid oxygen

*C.M.H. Ramage, S.S. Kee and A. Bristow*

395

Cyclopropane and the Datex Capnomac. Effect of cyclopropane on the single wavelength infrared measurement of volatile anaesthetic agents

*D.G. Mason and A.R. Lloyd-Thomas*

398

A coaxial technique for facilitating one-lung ventilation

*I.D. Conacher*

400

## FORUM

Wound infiltration of local anaesthetic after lower segment Caesarean section

*T.N. Trotter, P. Hayes-Gregson, S. Robinson, L. Cole, S. Coley and D. Fell*

404

Midazolam-droperidol premedication for cardiac surgery. A comparison with papaveretum and hyoscine

*J.H.L. Antrobus, P. Abbott, C.M.E. Carr and R.R. Chatrath*

407

Patients' desire for information about anaesthesia. Scottish and Canadian attitudes

*M. Lonsdale and G.L. Hutchison*

410

Flumazenil in intensive care. The duration of arousal after an assessment dose

*G.C. Fisher, M.C.C. Clapham and P. Hutton*

413

## CORRESPONDENCE

417

## BOOK REVIEWS

428



0003-2409(199105)46:5-G

12 1/2

# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 46 Number 6 June 1991

66

WCM



The Association of Anaesthetists of Great Britain and Ireland  
*Patron: HRH The Princess Margaret, Countess of Snowdon*



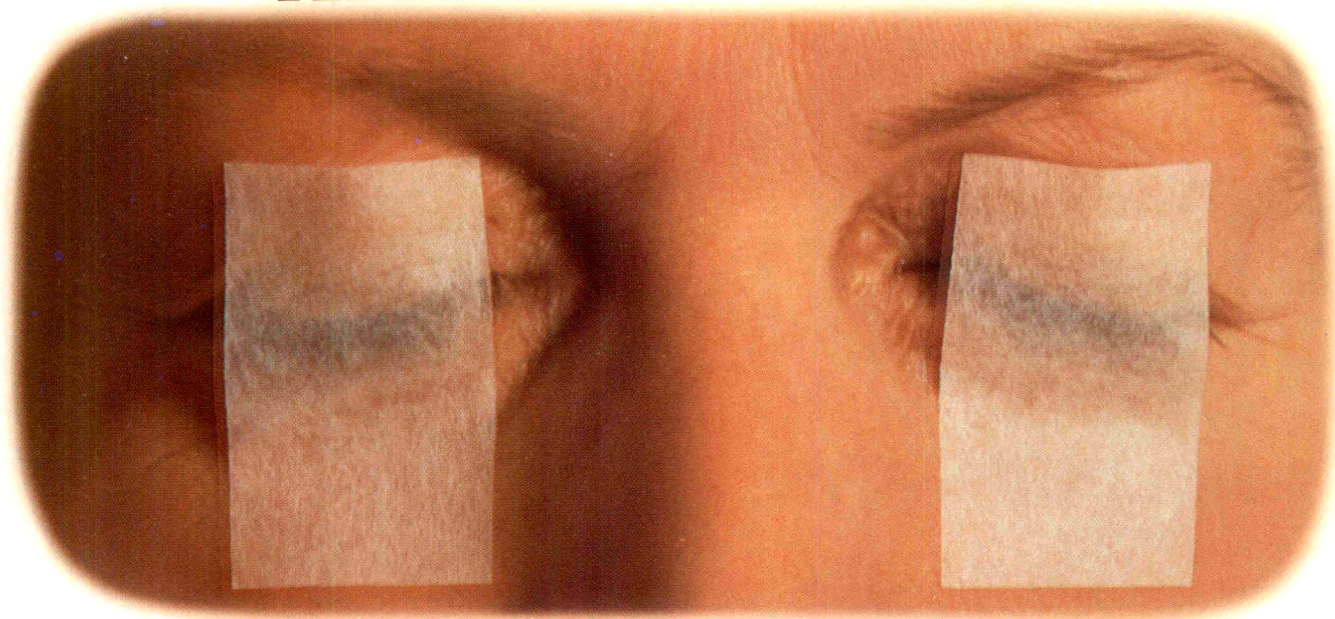
Published for the Association by  
Academic Press Grune & Stratton  
London San Diego New York Boston  
Sydney Tokyo Toronto



ISSN 0003-2409



# AFFECT THE EYE?



Basal tear production can decline by as much as 95% during general anaesthesia,<sup>1</sup> resulting in drying of the corneal epithelium and painful corneal abrasions.<sup>2</sup>

That's why it's so important to protect the cornea with LACRI-LUBE, the leading ophthalmic ointment for the treatment of dry eye.

LACRI-LUBE supplements deficient tears throughout a surgical procedure, helping to prevent accidental tape adhesion to the cornea or conjunctiva. In long procedures, further applications may be required.



And now LACRI-LUBE is available in a new **preservative-free** formulation which eliminates the potential for preservative sensitivities.

Yet another reason for you to choose LACRI-LUBE as an automatic part of your induction routine. After all, who needs eye problems after a hip operation?



## LACRI-LUBE\*

(preservative free ophthalmic ointment)

FOR CONTINUOUS CORNEAL PROTECTION DURING  
GENERAL ANAESTHESIA

### LACRI-LUBE\*

**Presentation:** Off-white, smooth, preservative-free sterile ophthalmic ointment containing white petrolatum, mineral oil and non-ionic lanolin derivatives. **Uses:** Useful as adjunctive therapy to lubricate and protect the eye in conditions such as exposure keratitis, decreased corneal sensitivity, recurrent corneal erosions, keratitis sicca, and also in ophthalmic and non-ophthalmic surgery. **Dosage and Administration:** For topical administration. Pull lower lid down to form pocket and apply a small amount as required. There is no variation of dose for age. **Contra-indications, warnings, etc.** No known contra-indications. **Pharmaceutical precautions:** Store away from heat. To avoid contamination during use, do not touch tube tip to any surface. **Legal Category:** P. **Package Quantities:** Lacri-Lube is available in 3.5g and 5g ophthalmic ointment tubes.

**Basic NHS cost:** 3.5g £1.97, 5g £2.49 (as at January 1991). **Further Information:** Dry eye symptoms commonly persist at night - Lacri-Lube has been specifically formulated to lubricate and protect the dry eye during sleep. Lacri-Lube can provide prophylactic ocular care during general surgical procedures as an adjunct to taping of the eyelids. **Product licence number:** 0426/0041

 **ALLERGAN PHARMACEUTICALS**

Allergan Ltd., High Wycombe, Bucks HP12 3SH.

**References:** 1. Cross, D.A. et al., *Anesthesia and Analgesia* ... *Curr Res* 1977; **56** (1): 35-37. 2. Snow, J.C. et al., *Anesthesia and Analgesia* ... *Curr Res* 1975; **54** (4): 465-467.



# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

---

Editor M. MORGAN

Assistant Editors R. GREENBAUM, A. R. AITKENHEAD, D. E. F. NEWTON, R. A. MASON, M. HARMER

## *Editorial Board*

Chairman The President of the Association P. J. F. BASKETT

Members A. M. ROLLIN, C. A. B. MCLAREN and a representative of the Junior Anaesthetists' Group

## *Ex officio*

The Honorary Treasurer of the Association W. R. MACRAE

The Honorary Secretary of the Association J. E. CHARLTON

The Immediate Past Honorary Secretary of the Association W. L. M. BAIRD

---

DIANE WINTERBURN, JANET SPRINGER, Reference librarians

---

*Address for literary contributions* Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK [telephone 081-740 3290].

Neither the Editor nor the Association of Anaesthetists of Great Britain and Ireland is in any way responsible for the statements made or the views expressed by contributors.

Investigations performed on man must conform to appropriate ethical standards including voluntary, informed consent and acceptance by an ethics committee.

A written statement of this acceptance may be requested by the Editor.

---

ANAESTHESIA: ISSN 0003-2409, Volume 46 1991, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. **All advertising** enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293). Annual subscription price including postage: £132 UK and US \$244 overseas. Personal subscription rate including postage: \$99. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel: 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

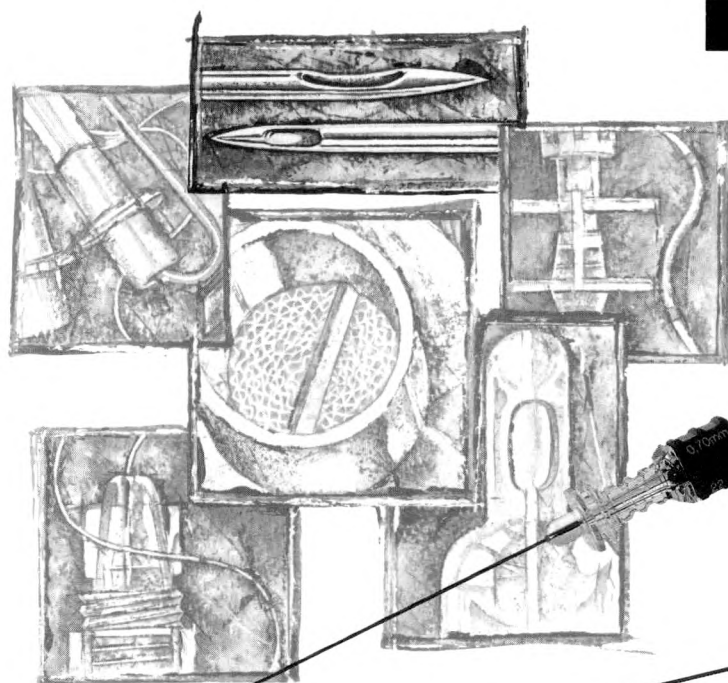
USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

*Anaesthesia* is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

© 1991 The Association of Anaesthetists of Great Britain and Ireland. The appearance of the code at the bottom of the first page of a paper in this journal indicates the copyright owner's consent that copies of the paper may be made for personal or internal use, or for the personal or internal use of specific clients in the USA. This consent is given on the condition, within the USA, that the copier pay the stated per-copy fee through the Copyright Clearance Center Inc., 27 Congress Street, Salem, MA 01970, USA for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to other kinds of copying, such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, for resale or for copying or distributing copies outside the USA.

Origination by BPCC Whitefriars Ltd, Tunbridge Wells, and printed and bound in Great Britain by BPCC Wheatons Ltd, Exeter.



## The Sprotte Spinal Needle

### The new atraumatic spinal needle

- Atraumatic tip displaces dural fibres.
- Dural tap closes on withdrawal of needle.
- Minimises loss of CSF and subsequent post dural puncture headaches.
- Easy injection and aspiration due to the relatively wide lateral eye.

21g, 22g, 24g

Sprotte spinal needles

now available from:

for further information write to:

RUSCH UK Ltd. Freepost, High Wycombe, Bucks. HP12 3BR or Freephone 0800 581121

**Rusch** U.K. Ltd.

10th WCA  
  
 World Congress of  
 Anaesthesiologists

The Hague, The Netherlands  
 12 - 19 June, 1992

For some must watch  
 while some must sleep

Shakespeare: Hamlet



Nederlandse  
 Vereniging  
 Anesthesiologie

# Call for papers

for the 10th World Congress  
 of Anaesthesiologists.

Deadline for abstracts:  
 1 November 1991

## Secretariat:

10th World Congress  
 of Anaesthesiologists

c/o Holland Organizing Centre  
 Lange Voorhout 16  
 2514 EE The Hague  
 The Netherlands

telephone (+31-70) 365.78.50  
 telex 33111 hoc nl  
 telefax (+31-70) 361.48.46

Application form for provisional programme, including abstract and registration forms. Please send back to the secretariat.

Name

Hospital/affiliation

Full mailing address

Country

Date

Signature

This preliminary application form entails no obligation whatsoever.



# HANDBOOK OF **Obstetric Anaesthesia**

**A. S. Buchan  
G. H. Sharwood - Smith**

This concise pocket book provides:

- *junior anaesthetists with a handy revision source for post graduate examinations*
- *qualified anaesthetists with a practical reference for the management of anaesthesia in obstetric emergencies*
- *obstetricians and midwives with an excellent guide to anaesthesia including adult and neonatal resuscitation*

Carefully selected reading lists are provided at the end of each chapter. The book also covers the immediate management of obstetric emergencies, for example:

- *major haemorrhage*
- *failed intubation*
- *eclampsia*
- *neonatal resuscitation*

Also covered are the management of medical problems in labour, the use of epidural analgesia and advice on the prevention and treatment of complications.

**A S Buchan MBChB FFARCS and G H Sharwood-Smith MBChB FFARCS are Consultants in Anaesthesia at the Simpson Memorial Maternity Pavilion, Royal Infirmary of Edinburgh.**

**0 7020 1540 7    April 1991    £9.95    128pp**

## **W. B. Saunders**

**Harcourt Brace Jovanovich, 24-28 Oval Road, London NW1 7DQ**





# Antifungal therapy that's severe on the infection, not on the patient.

Diflucan Oral/I.V. is a highly effective, well tolerated therapy with a convenient, once a day dosage and administration.

Overall clinical responses in deep-seated fungal infections are impressive: 87% in systemic candidiasis<sup>1</sup>, 96% in mucosal candidiasis<sup>2</sup> and 62% in cryptococcal meningitis.<sup>3</sup> Tolerability is similarly impressive with adverse effects comparable to placebo.<sup>4</sup>

Diflucan Oral/I.V. Treating invasive fungal infection with an iron fist and patients with a velvet glove.

**Diflucan** *ORAL/I.V.*  
fluconazole \*Trade Mark

*Counters invasive fungal infection*



## Editorial

### Purchasing equipment

The prospect of having a new suite of operating theatres and intensive care unit (ICU) is an exciting one. At last there will be the opportunity of replacing historical equipment and moving into a more attractive environment. This is particularly so if one has been through the planning process on more than one occasion, only to be let down at the last moment because there's no money. However, it also carries the danger of yielding to the pressure that is inevitably applied to buy the cheapest, so that you end up with what you don't want. We have recently been in the position of having to equip a new suite of operating theatres and an intensive care unit in a postgraduate, general teaching hospital. This 'high tech' monitoring was required in a number of areas to cope with cardiac and hepatobiliary surgery (although not transplantation) as well as the usual general, orthopaedic, paediatric and gynaecological surgery. We were determined not to fall into the trap of buying inappropriate equipment and our experiences in achieving this aim may well help others.

An Advisory Group was set up consisting of two consultant anaesthetists and a medical physics specialist. This group was aware that any project costing over £175 000 has to be advertised in European journals. They were further aware of the government's 'buy British' policy, with European manufacturers to be considered next;<sup>1</sup> purchase of equipment from the rest of the world is only to be considered if it is superior in design and performance, but the user must be prepared to justify his/her decisions under these circumstances.

The new operating suite consisted of eight theatres, eight anaesthetic rooms and a 12-bedded recovery area. There were eight beds in the ICU. We were inevitably asked what equipment we could take with us. Particularly important in this context is the European Communities Council Directive on Product Liability,<sup>2</sup> which passed into UK law in 1988. Attention is drawn to Article 11 of this directive, which automatically extinguishes the right to proceedings against a manufacturer for product liability 10 years after the date on which the producer put the product into circulation. As a result of this, we identified the need for eight new anaesthetic machines and 16 ventilators in the operating theatres. Five new ventilators were required for the intensive care unit, while new monitoring equipment was required in all areas. We discovered that if we bought from the manufacturers of our existing equipment, even though the models were different, we would avoid the delays and difficulties encountered in going out to tender. This policy was accepted and the supplies department handled the financial details of buying the new anaesthetic machines. We also perceived at this stage that combining the use of equipment less than 10 years old with the need for the purchase of new products would start our programme of planned replacement.

Our main need therefore was to buy monitoring equipment. It was at this stage that we discovered that £100 000 had arbitrarily been allocated by the planning

team for patient monitoring in the 28 operating theatre and recovery locations. No information was available about the amount of money required for monitoring equipment in operating theatres and the figures had been based on the £125 000 documented for central monitoring in an ICU. The planning team were informed that this sum was totally inadequate.

After discussion, the Advisory Group were requested to prepare a detailed specification of an integrated modular system, based on minimum monitoring requirements. The latter was defined as noninvasive blood pressure, pulse oximetry, capnography and an ECG at each of the 22 stations. In addition, 14 areas were defined as 'high tech' which required invasive pressure monitoring (2-3 channels), together with desirable features of cardiac output, temperature and ventilatory functions monitoring. We were determined to avoid the dangers of choosing equipment which was more complex and costly than required. This was especially important with regard to the anaesthetic machines; we decided that we only wanted devices that monitored machine function.

The document, Health Equipment Information (HEI) 98,<sup>3</sup> lays down clearly defined procedures for selection of products which are of good quality and adequate performance. It states that the responsibility for the final selection of equipment is vested in the user. This was particularly pertinent in view of the fact that a Regional standardisation policy existed. Adherence to the advice in HEI 98 proved to be invaluable.

We defined the functional requirement of the equipment to be purchased in as much detail as possible and considered the five basic components of the monitoring process,<sup>4</sup> namely signal generation and its transmission, processing, presentation and display. The essential and desirable features relating to analogue or digital displays of a 'user friendly' nature were defined. We sought total continuity of patient monitoring between the anaesthetic, operating and recovery rooms and the ICU, with battery back up in case of electrical failure, and which would also be required for interhospital transfer. A battery operated transport LCD module was also specified. We specified a network capable of allowing transmission of physiological data from some operating and recovery areas to ICU and to two central stations with printout facilities. We specified that we did not want a central nursing station in the ICU. The cost of installation, maintenance and user training was requested. We also required adequate manufacturing technical support in the UK and the cost of spares and consumables. The range of products were demonstrated on site, allowing discussions with the Anaesthetic Department and other user groups. Detailed specifications were obtained from the manufacturers at this stage. Tenders were then requested.

Receipt of the spread sheets from the manufacturers demonstrated the value of close scrutiny, which highlighted alarming discrepancies in some between the

specifications requested and those quoted. Failure to have done this could have resulted in major embarrassment. Some manufacturers even quoted for a central nursing station in the ICU. The submissions were not always for the correct number of beds and incomplete lists of equipment were also offered; pulse oximeters, temperature, additional pressure monitoring and capnography were frequently omitted. Differences in presentation of data from those requested, cramped screen displays with signal overlap and omission of freeze capability were identified. Network costs were sometimes not quoted, which would have been a significant hidden extra at £1200 per bed.

The two firms who most closely matched our specifications were invited to attend for discussions under the auspices of the supplies department to clarify their tenders. At no time were the clinicians involved in questions of finance. On the basis of the evidence presented, the General Manager agreed to the recommendations of the Advisory Group and allowed negotiations to proceed on the basis of procuring the right equipment for the hospital and not on the question of cost or country of origin.

We now have the monitoring equipment that we wanted in the areas we wanted it. Following the recommendations of HEI98 (updated November 1990) helped us to ensure that we achieved this aim. The Association's publication<sup>5</sup> on minimum monitoring is a

powerful tool in negotiation with management. Good maintenance procedures and definition of a replacement programme are in place, but whether funding will be available for the latter when necessary is still open to doubt.

*Department of Anaesthetics,  
Hammersmith Hospital,  
Du Cane Road,  
London W12 0HS*

J. LUMLEY  
D.A. ZIDEMAN

#### References

1. Cabinet Office. Advisory Council for Applied Research and Development. Medical Equipment 1986.
2. Official Journal of the European Communities No. L210. Council directive on 25 July 1985 on the approximation of the laws, regulations and administrative provisions of Member States concerning liability for defective products 1985, 29-33.
3. Health Equipment Information No. 98 Management of Equipment 1982. Issued by the Department of Health and Social Security and the Welsh Office.
4. HOPE CE, MORRISON DL. Understanding and selecting monitoring equipment in anaesthesia and intensive care. *Canadian Anaesthetists' Society Journal* 1986; 33: 670-9.
5. Recommendations for standards of monitoring during anaesthesia and recovery. Association of Anaesthetists of Great Britain and Ireland, 1988.

#### Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; 1: 432 5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

## Incidence of awareness with recall during general anaesthesia

W. H. D. LIU, T. A. S. THORP, S. G. GRAHAM AND A. R. AITKENHEAD

### Summary

*One thousand patients who were anaesthetised between February and April 1990 at University Hospital, Nottingham were interviewed between 20 and 36 hours after their operation. Patients under 16 years of age, those who had undergone obstetric or intracranial surgery, those who were unable to communicate and patients who were discharged from hospital before the postoperative visit were not interviewed. A standard set of questions was used to determine the incidences of recall of events and dreams during the operation. These incidences were 0.2% and 0.9% respectively, considerably lower than reported in previous comparable studies.*

### Key words

*Complications; awareness.*

The problem of awareness with recall during general anaesthesia has always been of great concern to both patients and anaesthetists. The psychological sequelae of subsequent recall of intra-operative events have been highlighted recently,<sup>1</sup> but the true incidence of awareness with recall, with or without prompting, is uncertain. There has been widespread publicity in recent years about awareness in obstetric anaesthetic practice; however, obstetric patients represent less than 30% of those who seek compensation for alleged awareness during general anaesthesia.<sup>2</sup> The last major study on this subject using a structured post-operative interview was in 1975.<sup>3</sup> Since then many new drugs have been introduced into anaesthetic practice, and techniques may also have changed as a result of media attention and increased consciousness of the problem by anaesthetists. The purpose of this study was to quantify the incidences of awareness with recall, and dreams, associated with current nonobstetric anaesthetic practice.

### Methods

All patients who were anaesthetised between February and April 1990 at University Hospital, Nottingham were identified. Categories of patient who were not interviewed are shown in Table 1. All other patients were interviewed between 20 and 36 hours after their operation by one of the first three authors using a standard set of questions shown in Table 2. Patients giving a history suggestive of awareness were subsequently reviewed by the fourth author to

**Table 1.** Categories of patients not interviewed in this study.

---

Patients under 16 years of age.  
Obstetric or intracranial surgery.  
Mental confusion.  
Patients unable to communicate in English.  
Patients who were discharged before the postoperative visit.

---

confirm the events. Awareness in this study was defined by the ability of the patients to recall, with or without prompting, any event which occurred between induction of anaesthesia and recovery of consciousness at the end of anaesthesia. Patients were not told the purpose of the visit until after the interview. Immediately after the interview the anaesthetic records were scrutinised and the drugs, techniques and any comments made by the anaesthetists recorded. None of the anaesthetists in the hospital, other than the authors, knew that the study was being undertaken. This was to ensure that their usual anaesthetic practice was not altered.

**Table 2.** Questions asked during structured interview.

- 
1. What was the last thing you remember before you went to sleep for your operation?
  2. What was the first thing you remember after your operation?
  3. Can you remember anything in between these two periods?
  4. Did you dream during your operation?
  5. What was the worst thing about your operation?
- 

W.H.D. Liu, MB, BCh, FCAnaes, Research Fellow, T.A.S. Thorp, MB, ChB, FFARCSI, Senior Registrar, S.G. Graham, MB, ChB, FCAnaes, Lecturer, A.R. Aitkenhead, BSc, MD, FCAnaes, Professor, University Department of Anaesthesia, University Hospital, Queen's Medical Centre, Nottingham NG7 2UH.

This paper is based on a presentation to the Anaesthetic Research Society in July 1990.

Accepted 8 October 1990.

**Table 3.** Types and frequency distribution of premedication.

Benzodiazepines	330
Opioid + hyoscine	187
Opioid + antiemetic	88
Opioid + other sedative	21
Others	3

### Results

One thousand patients were interviewed between February and April 1990. Sedative premedication was given to 629 patients (62.9%) (Table 3); 316 patients breathed spontaneously throughout the procedure, 92 received suxamethonium to facilitate tracheal intubation only and breathed spontaneously thereafter, and 592 received intermittent positive pressure ventilation throughout surgery. In the last group, 19 patients received suxamethonium and the remainder nondepolarising muscle relaxants.

The age and sex distributions of the patients are shown in Figure 1. There was a preponderance of females aged 36–55 years, the majority of whom were undergoing gynaecological procedures. Table 4 shows the types and number of operations.

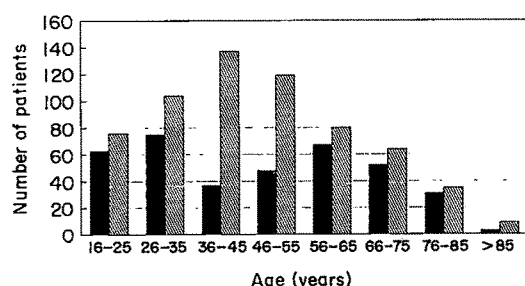
Two patients (0.2%, 95% confidence intervals 0.02–0.7%) were found to have been aware and recalled events shortly after the induction of anaesthesia, but before the start of surgery.

#### Case history 1

A 35-year-old man, ASA 2, weighing 82 kg, presented for tonsillectomy. He was premedicated with papaveretum 20 mg and hyoscine 0.4 mg. Anaesthesia was induced with methohexitone 100 mg. His trachea was intubated after suxamethonium 100 mg had been given and anaesthesia was maintained with nitrous oxide 67% in oxygen, and halothane 2%. The halothane was substituted by isoflurane when ventricular extrasystoles occurred. This patient recalled laryngoscopy and tracheal intubation, but apart from the presence of ventricular extrasystoles, there were no obvious clinical signs which suggested that the patient might have been aware. This patient had been aware of intra-operative events during an appendicectomy at 13 years of age, but this was not elicited by the pre-operative assessment for the present operation.

#### Case history 2

A 73-year-old woman, ASA 2, weighing 102 kg presented for repair of an incisional hernia. She was premedicated



**Fig. 1.** Distribution of the age and sex of the patients interviewed. ■, males; ▨, females.

**Table 4.** Types and number of operations. 'Other surgery' refers to noncranial surgery performed by neurosurgeons.

General surgery	324
Gynaecology	298
Orthopaedics	160
Ear, nose and throat	113
Ophthalmology	97
Others	8

with temazepam 10 mg. Anaesthesia was induced with thiopentone 200 mg and her trachea intubated after the administration of vecuronium 7 mg. She was then given fentanyl 50 µg, and anaesthesia was maintained with 50% nitrous oxide in oxygen in the anaesthetic room. Systolic arterial blood pressure was 200 mmHg and heart rate 110 beats/minute when monitoring started in the operating theatre. Administration of enflurane 0.5% was then started. She had no recall of tracheal intubation, but remembered being wheeled into the operating theatre from the anaesthetic room and being placed on to the operating table. Neither of these patients recalled any part of the operation, nor were they unduly distressed by their experiences.

A further nine patients (0.9%, 95% confidence intervals 0.41–1.71%) reported having had dreams during anaesthesia; none of these patients reported awareness. None of the dreams was unpleasant and none was related in any way to intra-operative events. Six of these patients received thiopentone for induction, two had propofol and one had etomidate.

Five patients reported awareness with recall during previous operations. Our structured interview did not include questions about previous operations: these patients 'volunteered' the information probably because leading questions were used about their present operation. Three of these cases of awareness with recall were not identified during the pre-operative visit for the present operation; two had been aware during obstetric procedures. Only one of these five patients was aware during this survey (case history 1).

### Discussion

Patients who undergo general anaesthesia expect to remember none of the events between induction of anaesthesia and recovery of consciousness after completion of surgery.<sup>1</sup> Conscious awareness with recall has always been of major concern to both patients and anaesthetists and can, particularly when pain is experienced, lead to serious psychological sequelae characterised by a form of neurosis. The latter may include insomnia, anxiety, depression, irritability, repetitive nightmares and a pre-occupation with death, and may lead to a phobia of hospitals and doctors.<sup>4-6</sup> In addition, from the point of view of anaesthetists, conscious awareness with recall is a major medicolegal problem.<sup>1,12</sup>

The most important aspect of awareness is what the patient can remember consciously, so it has been suggested that the most appropriate method to investigate the phenomenon of conscious awareness with recall is to conduct a structured interview in the postoperative period.<sup>7</sup> The actual timing of this type of interview is a matter of debate. Some advocate that patients should be interviewed as soon as they regain consciousness, for example in the recovery

**Table 5.** Summary of incidences of awareness with recall and dreaming in studies using a structured interview.

Author	Date	Awareness (%)	Dreaming (%)	Sample size
Hutchinson <sup>10</sup>	1960	1.2	3.0	656
Harris <sup>12</sup>	1971	1.6	26.0	120
McKenna <sup>13</sup>	1973	1.5	—	200
Wilson <sup>3</sup>	1975	0.8	7.7	490
Present study	1990	0.2	0.9	1000

room. However, the majority of patients will still be drowsy and may therefore give an unreliable account. Furthermore, the confidentiality of this type of survey may be difficult to maintain. Others suggest that patients should be interviewed much later, for instance one week after the operation,<sup>8</sup> so that they may have more time to reflect upon the sequence of events. This would result in failure to interview patients already discharged from hospital and therefore may not be representative. A compromise was therefore made in this study to interview patients 20 to 36 hours after their operation.

Patients who had undergone obstetric operations were excluded because it is possible that such operations have an increased incidence of awareness with recall.<sup>9</sup> Other groups of patients were excluded (Table 1) because of difficulties in communication and/or unreliable history.

The incidence of conscious awareness with recall in this study was 0.2%. Table 5 compares our results with those of the four previous comparable studies published over the last 30 years in nonobstetric anaesthesia (i.e. those in which a postoperative structured interview was employed). There is a suggestion that the incidence of awareness with recall has decreased over the past 15 years, although these studies are not directly comparable. This apparent reduction in incidence is probably the result of heightened publicity by the media and increased awareness of the problem by anaesthetists. Both our patients who had awareness with recall remembered events during the period shortly after induction of anaesthesia but before the start of surgery.

Furthermore, the incidence of dreams appeared to be much lower in our study compared with previous reports.<sup>10,11</sup> The relationship between dreams and light anaesthesia is not fully established,<sup>9</sup> and while every effort was made to ensure, by close questioning, that those patients who said they had dreamt actually dreamed during the operation, it is possible that some had dreamt in the immediate postoperative period and not during surgery. There is no way to confirm this. It has been suggested that dreaming is more likely if certain types of anaesthetic agent are used but, partly because of the low incidence of dreams in this study, there was no clear relationship between the occurrence of dreams and the type of anaesthetic agents.

The commonest reason for conscious awareness with recall is faulty anaesthetic technique.<sup>2</sup> This was probably true in our two patients with awareness; both had received a relatively small induction dose of anaesthetic drugs. Most cases of awareness with recall may be preventable by meticulous technique, but it is unlikely that awareness in patients who are paralysed by muscle relaxants can be eliminated totally without unduly endangering some patients by administration of excessive amounts of anaesthetic agents.<sup>14</sup> It is clear that sedative premedication

cannot be relied upon to prevent awareness with recall since both of these patients were premedicated.

In conclusion, there is some evidence to suggest that the incidence of conscious awareness with recall and dreams has reduced in the last 15 years. This may have been because of increased consciousness of the problem by anaesthetists. It was also clear from our survey that routine pre-operative visits did not necessarily identify patients who have had awareness with recall during their previous operations; this may suggest that leading but tactful questions about previous anaesthetics may be helpful in identifying those patients who have been aware, since these patients may require additional reassurance and counselling. In addition, previous awareness may indicate decreased susceptibility to the effects of anaesthetic agents. None of our patients had recall of any events during surgery, but two remembered events shortly after induction of anaesthesia. This suggests that anaesthetists should concentrate not only on the anaesthetic technique during the operative procedure, but also pay meticulous attention during the transitional phase from intravenous induction to inhalational maintenance.

#### Acknowledgment

This study was supported by a grant from the BUPA Medical Foundation Ltd.

#### References

1. AITKENHEAD AR. Awareness during anaesthesia: what should the patient be told? *Anaesthesia* 1990; **45**: 351–2.
2. HARGROVE RL. Awareness: a medicolegal problem. In: ROSEN M, LUNN JN, eds. *Consciousness, awareness and pain in general anaesthesia*. London: Butterworths, 1987: 149–54.
3. WILSON SL, VAUGHAN RW, STEPHEN CR. Awareness, dreams and hallucinations associated with general anaesthesia. *Anesthesia and Analgesia* 1975; **54**: 609–17.
4. MEYER BC, BLACHER RS. A traumatic neurotic reaction induced by succinylcholine chloride. *New York State Journal of Medicine* 1961; **61**: 1255–61.
5. BERGSTRÖM H, BERNSTEIN K. Psychic reactions after analgesia with nitrous oxide for caesarean section. *Lancet* 1968; **ii**: 541–2.
6. BLACHER RS. On awakening paralyzed during surgery: a syndrome of traumatic neurosis. *Journal of the American Medical Association* 1975; **234**: 67–8.
7. JELICIC M, BONKE B. The incidence of awareness during anaesthesia. *Anaesthesia* 1989; **44**: 1004–5.
8. ADAM N. Disruption of memory functions associated with general anaesthetics. In: KIHTSTROM JF, EVANS FJ, eds. *Functional disorders of memory*. Hillsdale, New Jersey: Lawrence Erlbaum Associates, 1979: 219–38.
9. BOGOD DG, ORTON JK, YAU HM, OH TE. Detecting awareness during general anaesthetic caesarean section: an evaluation of two methods. *Anaesthesia* 1990; **45**: 279–84.
10. HUTCHINSON R. Awareness during surgery: a study of its incidence. *British Journal of Anaesthesia* 1961; **33**: 463–9.
11. BRICE DD, HETHERINGTON RR, UTING JE. A simple study of awareness and dreaming during anaesthesia. *British Journal of Anaesthesia* 1970; **42**: 535–41.
12. HARRIS TJB, BRICE DD, HETHERINGTON RR, UTING JE. Dreaming associated with anaesthesia: the influence of morphine premedication and two volatile adjuvants. *British Journal of Anaesthesia* 1971; **43**: 172–8.
13. MCKENNA T, WILTON TNP. Awareness during endotracheal intubation. *Anaesthesia* 1973; **28**: 599–602.
14. BLACHER RS. Awareness during surgery. *Anesthesiology* 1984; **61**: 1–2.

## Patient-controlled on-demand epidural fentanyl

A comparison of patient-controlled on-demand fentanyl delivered epidurally or intravenously

E. A. WELCHEW AND D. P. BREEN

### Summary

*A prospective, open, clinical trial is described in which 20 patients having upper abdominal surgery were randomly allocated to receive fentanyl for postoperative analgesia by patient-controlled demand analgesic computer by either the epidural or intravenous route. Hourly pain, sedation and nausea scores were very similar in the two groups during the first 24 hours after surgery. What few differences there were favoured the epidural group. There was a highly significant difference in fentanyl consumption between the two groups, with the intravenous group demanding consistently more than twice as much as the epidural group.*

### Key words

*Pain; postoperative.*

*Anaesthetic techniques; epidural, intravenous.*

*Analgesics; fentanyl.*

Severe postoperative pain has a well known morbidity and causes distress to patients.<sup>1-3</sup> Several surveys of both patients and hospital staff have suggested that postoperative pain is still poorly treated.<sup>4-6</sup> Despite the introduction of new analgesics, advances in pain relief are most likely to come from improvements in the delivery of existing drugs to the patient.

Within the last decade there have been two major advances in this field. The first was the introduction of spinal and epidural narcotics delivered locally to the 'target' spinal pain pathways.<sup>7,8</sup> The second was the introduction of patient-controlled, on-demand analgesic systems which allow patients to titrate the amount of analgesic they receive directly against the amount of pain they are feeling.<sup>9,10</sup>

Despite a wealth of clinical and experimental evidence to the contrary, there still appears to be a view that the delivery of narcotics epidurally is simply an invasive way of establishing a depot for absorption into the blood. If this were true, then a single dose of the drug given epidurally would have a slower onset, reduced peak effect and longer duration of action compared with the same dose given intravenously. If the drug were given by continuous infusion by the two different routes to achieve identical end-points, then once redistribution had settled down, the

infusion rates would simply match the clearance of the drug and would therefore be identical.

On-demand analgesic systems have been advocated as one of the ways in which microprocessors may be used to tailor the timing of analgesic delivery to patient requirements. They are able to provide patients with immediate access to an analgesic in small repeated doses whenever, and only when, they require it. These systems have been advocated for potency-duration studies of analgesics because patients set their own end-point for drug effects and titrate the total dose of analgesic against that end-point. The assumption inherent in this would be that the average end-point for a group of patients with similar operations would always be the same, despite wide inter-patient variations. Groups of patients who had the same operation could therefore be compared with other groups who had the same operation, but used a different drug in the on-demand system.

It was considered that an on-demand analgesic system could provide the opportunity for the study of the potency-duration of a narcotic when given by two different routes; in particular, the epidural and intravenous routes. On the basis that fentanyl has a predominantly local, spinal action, the hypothesis to be tested was that the rate of consumption of fentanyl would be significantly lower when the drug

E. A. Welchew, MB ChB, FCAnaes, Consultant, Department of Anaesthesia, Northern General Hospital, Herries Road, Sheffield S5 7AU, D. P. Breen, BMedSci, BM, BS, FCAnaes, Research Fellow, Intensive Care Unit, Honorary Research Assistant, University Department of Anaesthesia, Royal Hallamshire Hospital, Glossop Road, Sheffield S10 2JF.

Accepted 13 October 1990.

was delivered epidurally compared with the intravenous route.

### Method

The protocol for the trial was approved by the hospital ethics committee. Twenty patients scheduled for noncancer, upper abdominal surgery in the middle of the afternoon were admitted to the trial. The patients were healthy, ASA grades 1 or 2, receiving no medication and having no allergies. An equal number of males and females were included. All patients entering the trial had previously given written, informed consent to participate in the trial and to have a thoracic epidural catheter inserted during the anaesthetic for the relief of postoperative pain.

Each patient was seen before operation when the trial was explained and written consent was obtained. During the visit the patients were instructed in the use of the on-demand analgesic system (Janssen Scientific Instruments On-Demand Computer or ODAC) and the electronic linear visual analogue system.<sup>11</sup> Premedication was with oral diazepam 10 mg given 2 hours before surgery.

All patients were given a standard anaesthetic consisting of induction with a titrated dose of etomidate (range 10–18 mg). The trachea was intubated after 6 mg of pancuronium had taken effect and the lungs were artificially ventilated with 33% oxygen in 67% nitrous oxide and halothane (0.5%–1%) using a Cape-Waine ventilator. Patients of each sex were randomly allocated to either the epidural or intravenous group after induction of anaesthesia, so that there were an equal number of each sex in each group. Patients in the epidural group had a thoracic epidural catheter inserted at the T<sub>7</sub>–T<sub>8</sub> interspace. Five ml of 0.9% saline was flushed down the catheter during surgery. The halothane was discontinued just before the end of surgery in both groups and residual neuromuscular blockade reversed with a mixture of atropine and neostigmine.

After surgery, patients in the epidural group had their epidural catheters connected to the ODAC via a 0.22 micron sterile filter (Millipore). Patients in the intravenous group had the ODAC connected to their intravenous fluid line at the catheter via a Cardiff nonreturn valve. In all cases the ODAC was primed with 200 ml of sterile saline solution containing 10 µg/ml of fentanyl. This concentration has been shown previously to be optimal for epidural fentanyl.<sup>12</sup> In each group, when the first demand for pain relief was made patients were given 10 ml of this solution either epidurally or intravenously, as appropriate. After this modest loading dose those in the epidural group obtained doses of 0.5 ml of the fentanyl in saline solution in response to each demand made to the ODAC, whilst those in the intravenous group obtained 2 ml of the same solution at each demand. The lockout interval of the ODAC system, during which no drug was administered to the patient in response to their demand, was set at 2 minutes. In addition to these demanded doses of analgesic, both groups also had a low-level continuous infusion of 20 µg of fentanyl/hour via each groups' respective route. The ODAC system was used continuously for at least 24 hours in each patient and observations made during the first 24 hours after surgery.

The patients made continuous observations on an electronic linear visual analogue system for pain, sedation and nausea. These observations were recorded on a chart

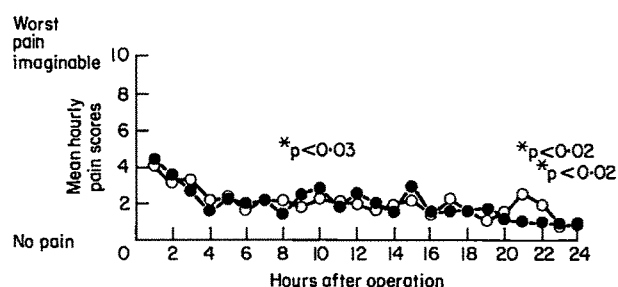


Fig. 1. Mean hourly pain scores. ●—●, epidural fentanyl; ○—○, intravenous fentanyl.

recorder with the results of a simple reaction test every 30 minutes. The observations were analysed blind in a standard manner in pairs to give the hourly pain, sedation and nausea scores, which had been corrected for periods of sleep.<sup>11</sup>

The ODAC recorded the times of each demanded dose of analgesic for each patient and also printed the cumulative dose for each patient with time. From this information, graphs of the mean cumulative dose of analgesic and mean hourly consumption of analgesic in each group could be drawn. The statistical significance in the consumption of fentanyl between the two groups was analysed using Student's *t*-test for unpaired data.

All linear analogue data were presented graphically as mean hourly scores; however, the statistical significance of the differences between the two groups was calculated using the Mann-Whitney *U* test. All other data in the study were tested using Student's *t*-test for unpaired data. Differences between the two groups were considered significant if  $p < 0.05$ .

### Results

Table 1 shows the mean age and weight for patients in the epidural and intravenous groups. The mean age of patients in the epidural group was just under 48 years whilst that of the intravenous was just under 43 years. This difference was not statistically significant. The difference in the mean weights of the patients in the two groups was also not statistically significant. Each group consisted of five men and five women and all data collected during the trial were included in the results with no patients failing to complete the trial.

Figure 1 shows the mean hourly pain scores of patients in the two groups during the whole 24 hours of observation. These data were collected from immediately after each patient received their 100-µg bolus dose of epidural or intravenous fentanyl. It shows that the pain scores at the end of the first hour were very similar in the two groups. Both groups showed a rapid decline in pain scores in the first 4 hours to a plateau which both groups sustained for

Table 1. Mean ages and weights of patients in the two groups.

Group	Mean ages (SD) years	Mean weights (SD) kg
Epidural	47.9 (14.99)	60.7 (11.60)
Intravenous	42.9 (8.10)	62.7 (8.91)



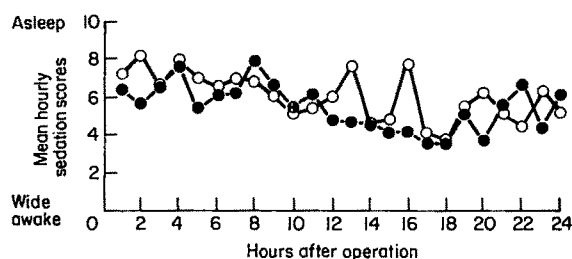


Fig. 2. Mean hourly sedation scores. ●—●, epidural fentanyl; ○—○, intravenous fentanyl.

the rest of the 24 hours of the study. The epidural group had significantly lower hourly pain scores than the intravenous group at 8, 21 and 22 hours after the start of the trial (Fig. 1). At no stage were the pain scores of the intravenous group significantly better than those of the epidural group.

Figure 2 shows how both groups had high initial sedation scores which slowly declined with time after their anaesthetics. The relatively wide scatter on this graph, particularly in the intravenous group, is probably the effect of random short periods of sleep, which is typical of the early postoperative patient. There were no statistically significant differences in hourly sedation scores between the two groups at any time during the period of study.

Mean hourly nausea scores are shown in Figure 3 which demonstrates that there were no statistically significant differences in the groups' hourly nausea scores, although the intravenous group did have a significantly higher aggregate nausea score for the first 12 hours of the study compared with the epidural group ( $p < 0.05$ ).

Table 2 shows the mean rates of fentanyl consumption in the two groups, averaged over the whole 24 hours of the study. It can be seen that the epidural group consumed less than half the dose of fentanyl consumed by the intravenous group. Patients in the epidural group also had a much narrower range of hourly consumptions of fentanyl than those receiving the drug intravenously. This difference in variability is also shown by the much reduced standard deviation in those patients receiving the drug epidurally.

Figure 4 displays the mean cumulative doses of fentanyl consumed by patients in each group. The differences between the groups are significant for every hour of the study.

Figure 5 shows the same information in a slightly different form, as mean hourly consumption of the drug. This graph also illustrates the high mean consumption during the first 2 hours of the study, after which both

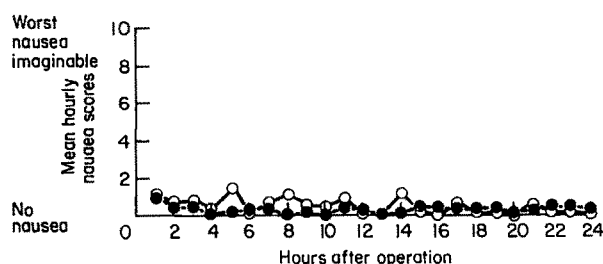


Fig. 3. Mean hourly nausea scores. ●—●, epidural fentanyl; ○—○, intravenous fentanyl.

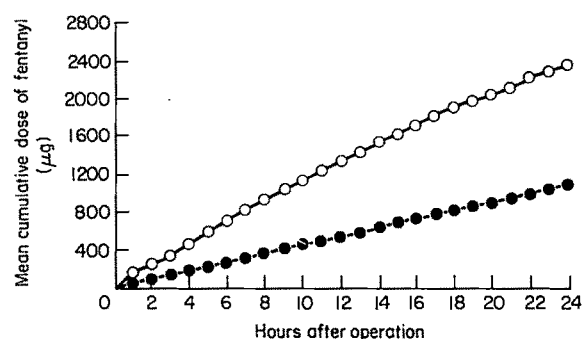


Fig. 4. Mean cumulative doses of fentanyl consumed by patients. ●—●, epidural fentanyl; ○—○, intravenous fentanyl.

groups settled into their individual patterns of consumption. This figure demonstrates the marked stability in the consumption of fentanyl shown by patients receiving the drug epidurally compared with those receiving the drug intravenously.

### Discussion

All of the patients in this study had upper right or left paramedian incisions for upper abdominal surgery performed by the same firm of surgeons at approximately the same time of day. Patients were nursed on the same pair of adjacent wards and their care was closely supervised by the authors during the first 24 hours after surgery. Each group consisted of equal numbers of males and females whose mean ages and mean weights were not significantly different. The mean pain scores of the two groups were very similar during the 24-hour period of observation. The only significant differences were in favour of the epidural route. Pain scores obtained from patients in this trial were generally low, compared with those of comparable patients reported previously.<sup>13,14</sup> There were no significant differences in the mean hourly sedation scores between the two groups and the only difference in the nausea scores was a significantly increased aggregate score for the first 12 hours of the study in the intravenous group. This suggests a small but significant increase in nausea during this time in this group. These differences in observations were associated with a highly significant reduction in the consumption of fentanyl in those receiving it epidurally.

The results of this trial demonstrated a clear increase in the potency-duration of fentanyl for analgesia when the drug was given epidurally, compared with the intravenous route. Epidural fentanyl was shown to have a potency-duration of at least 2.2 times that of the same drug given intravenously. The only possible explanation for this is that the majority of the analgesic effect of fentanyl delivered epidurally must be locally mediated and these effects of the drug were not the result of systemic absorption. On the

Table 2. Mean rates and range of fentanyl consumption in the two groups.

Group	Mean (SD) µg/hour	Range µg/hour
Epidural	44.04 (14.92)	20–130
Intravenous	98.17 (51.17)	10–290

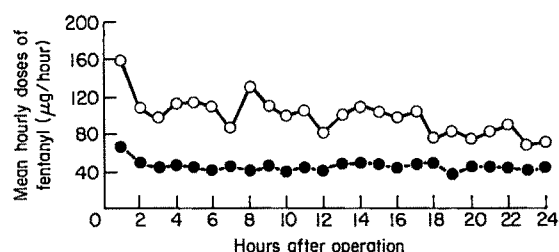


Fig. 5. Mean hourly consumption of fentanyl by patients. ●—●, epidural fentanyl; ○—○, intravenous fentanyl.

other hand, nausea scores were significantly increased in those patients receiving the largest total dose of fentanyl, which suggests that in these patients, nausea may have been mediated by fentanyl in the blood.

In conclusion, this prospective, open trial has demonstrated that when using a patient-controlled on-demand analgesic system, patients having fentanyl delivered epidurally had slightly better analgesia than those receiving the same drug intravenously, despite consuming less than half as much fentanyl during the same period. This suggests that the majority of the analgesic effect provided by the epidural fentanyl is locally mediated within the vertebral canal, whilst side effects such as nausea may be related to uptake of the drug into the blood.

#### Acknowledgments

We thank Ms J. Hosking SRN (Analgesic Sister) for her invaluable assistance in looking after the patients admitted to the trial. We also thank Professor A.G. Johnson and Mr J.A.R. Smith for permission to study patients under their care. We are indebted to Janssen Pharmaceutical Limited for loan of the on-demand analgesic computer (ODAC) used in this trial.

#### References

1. WIGHTMAN JAK. A prospective survey of the incidence of postoperative pulmonary complications. *British Journal of Surgery* 1968; **55**: 85–91.
2. PFLUG AE, BONICA JJ. Physiopathology and control of post-operative pain. *Archives of Surgery* 1977; **112**: 773–81.
3. SPENCE AA. Relieving acute pain. *British Journal of Anaesthesia* 1980; **52**: 245–6.
4. COHEN FL. Post surgical pain relief: patients' status and nurses' medication choices. *Pain* 1980; **9**: 265–74.
5. MARKS RM, SACHAR EJ. Undertreatment of medical inpatients with narcotic analgesics. *Annals of Internal Medicine* 1973; **78**: 173–81.
6. WEISS OF, SRIWATANAKUL K, ALLOZA JL, WEINTRAUB M, LASAGNA L. Attitudes of patients, housestaff and nurses towards postoperative analgesic care. *Anaesthesia and Analgesia* 1983; **62**: 70–4.
7. WANG, JK, NAUSS LA, THOMAS JE. Pain relief by intrathecally applied morphine in man. *Anesthesiology* 1979; **50**: 149–51.
8. BEHAR H, OLSHWANG D, MAGORA F, DAVIDSON JT. Epidural morphine in treatment of pain. *Lancet* 1979; **1**: 527–8.
9. TAMSEN A, HARTVIG B, DAHLSTRÖM B, LINDSTRÖM B, HOLMDAHL MH. Patient controlled analgesic therapy in the early postoperative period. *Acta Anaesthesiologica Scandinavica* 1979; **23**: 462–70.
10. WHITE WD, PEARCE DJ, NORMAN J. Postoperative analgesia: a comparison of intravenous on-demand fentanyl with epidural bupivacaine. *British Medical Journal* 1979; **2**: 166–7.
11. WELCHEW EA. A postoperative pain recorder. A patient-controlled recording device for assessing postoperative pain. *Anaesthesia* 1982; **37**: 838–41.
12. WELCHEW EA. The optimum concentration for epidural fentanyl. A randomised, double-blind comparison with and without 1:200 000 adrenaline. *Anaesthesia* 1983; **38**: 1037–41.
13. WELCHEW EA, THORNTON JA. Continuous thoracic epidural fentanyl. A comparison of epidural fentanyl with intramuscular papaveretum for postoperative pain. *Anaesthesia* 1982; **37**: 309–16.
14. WELCHEW EA. On-demand analgesia. A double-blind comparison of on-demand intravenous fentanyl with regular intramuscular morphine. *Anaesthesia* 1983; **38**: 19–25.

## A comparison of APACHE II and a clinical sickness score

A study of 97 consecutive admissions to a District General Hospital Intensive Care Unit

J. R. SINCLAIR, P. T. MAGEE, T. H. GOULD AND C. H. COLLINS

### Summary

*The aim of this study was to compare the predictive power of a simple illness severity score (Clinical Sickness Score) to that of APACHE II in a District General Hospital intensive therapy unit. A prospective comparison was carried out on 97 consecutive adult patients whose severity of illness was scored one hour after admission using both the Clinical Sickness Score and APACHE II. Intensive Therapy Unit and hospital outcomes were recorded for each patient. The Clinical Sickness Score and APACHE II identified survivors and nonsurvivors with similar power ( $p < 0.001$ ). There was a highly significant correlation between the two scoring systems for hospital survivors and nonsurvivors together ( $r = 0.5418$ ,  $r^2 = 0.28$ ,  $p = < 0.0001$ ) and for hospital survivors alone ( $r = 0.6102$ ,  $r^2 = 0.37$ ,  $p = 0.0001$ ). Correlation for hospital nonsurvivors was not significant ( $r = 0.1629$ ,  $r^2 = 0.027$ ,  $p = 0.3134$ ). The positive predictive values of APACHE II were between 5% and 10% more sensitive than the Clinical Sickness Score for hospital outcome. Admission Clinical Sickness Score and APACHE II scores had similar predictive power in this study.*

### Key words

*Intensive care; illness severity scoring.*

Illness severity scores provide a means of comparing the results of treatment, evaluating changes in therapy, and achieving consistent standards. The Acute Physiology and Chronic Health Evaluation II score (APACHE II)<sup>1</sup> is becoming an established scoring system in intensive care practice, and 1990 saw the completion of data collection in the Intensive Care Society's multicentre UK APACHE II Study. Many workers have attempted to validate APACHE II since Knaus' original work<sup>2-4</sup> using prediction of outcome as a measure, and there are studies comparing APACHE II with other illness severity scores.<sup>5</sup>

However, it is not certain that APACHE II is an appropriate illness severity score for district general hospital intensive therapy unit (ITU) audit. APACHE II is time-consuming and complex, and might be open to inaccuracy as a result, particularly in busy units. The score is made up of a physiological score, weighted for age, diagnostic group and chronic health. A simplified illness severity score, the Clinical Sickness Score (CSS), was developed in Central Africa in 1986 and provided an objective measurement of illness severity for 624 African ITU patients.<sup>6</sup> It is essentially the Glasgow Coma Score with cardiorespiratory and temperature scores, and a weighting for age. The score weighting, where appropriate, is based on that used in APACHE II and the complete system is shown in Appendix 1.

The aim of this study was to compare the predictive power of the CSS with that of APACHE II. Any evidence that this scoring system was as accurate as APACHE II in predicting hospital outcome from intensive care would represent an advance in audit, as it is simpler, less time consuming, and potentially more reproducible than APACHE II.

### Methods

The Royal Devon and Exeter Hospital is a busy district general hospital. Its ITU has six beds and is staffed by resident anaesthetic registrars supported by consultants

Table 1. Characteristics of the 97 patients studied.

Male: female	60:37
Median (range) age; years	57 (20-87)
<i>Major organ system categories*</i>	
Cardiovascular	26
Respiratory	29
Gastrointestinal	10
Neurological	14
Renal	16
Haematological	2

\* Adapted from Knaus.<sup>1</sup>

J.R. Sinclair, FCAnaes, P.T. Magee, FCAnaes, Senior Registrars, C.H. Collins, FCAnaes, Consultant, Royal Devon and Exeter Hospital, Exeter EX2 5DW, T.H. Gould, MRCP, SHO, Frenchay Hospital, Bristol BS16 1LE.

Accepted 11 October 1990.

**Table 2.** Results of Mann-Whitney *U* test comparing survivors and nonsurvivors scored with CSS and APACHE II in relation to ITU and hospital outcome.

Group	Survivors, median score (range)	Nonsurvivors, median score (range)	U value	Z statistic	p
CSS ITU outcome	8 (1-27)	18 (3-26)	485	-4.3546	< 0.001
CSS Hospital outcome	8 (1-27)	18 (3-26)	641	-3.6109	< 0.001
APACHE II ITU outcome	14 (1-29)	22 (7-45)	410	-4.9237	< 0.001
APACHE II Hospital outcome	13 (1-34)	22 (7-45)	372	-5.5913	< 0.001

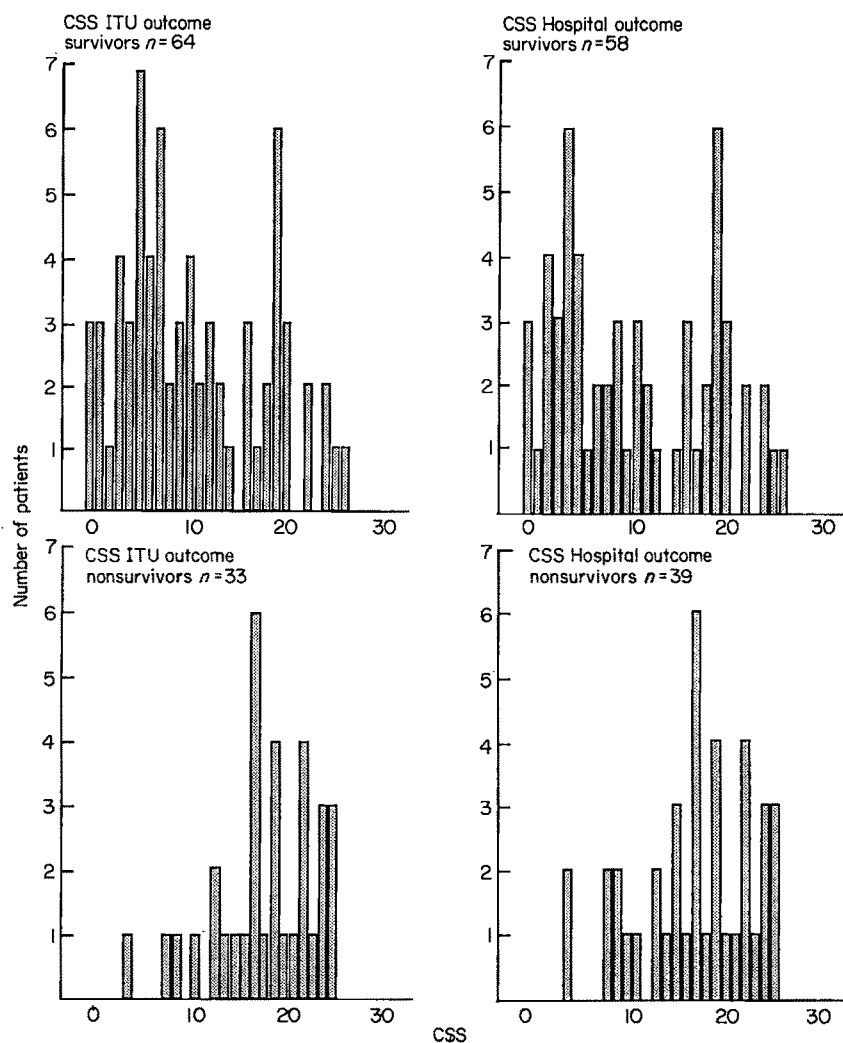
who undertake daily rounds. Patients are admitted from general, thoracic and vascular surgical units, general and renal medical beds, after orthopaedic and trauma surgery and from a subregional haematology unit.

Ninety-seven consecutive patients were scored by two authors one hour after admission to ITU. Each patient received two scores, (one CSS and one APACHE II) and these scores were recorded with the patient's age, sex, diagnosis and clinical summary. We omitted age-weighting in the CSS in this study because chronological age has a different physiological consequence in Africa from that in

Europe. Each patient was followed throughout his or her hospital stay. The ITU and hospital outcomes (survivor or nonsurvivor) were recorded.

The null hypothesis for each group was that the survivors had no tendency to score differently from the nonsurvivors. The Mann-Whitney *U* test was used to analyse the results.

Death was selected as the 'outcome positive' in calculating the sensitivity (accuracy of prediction of survivors) and the specificity (accuracy of prediction of nonsurvivors) of both scoring systems. Scores greater or equal to both 18

**Fig. 1.** Clinical Sickness Scores (CSS) in survivors and nonsurvivors in respect of ITU and hospital outcome.

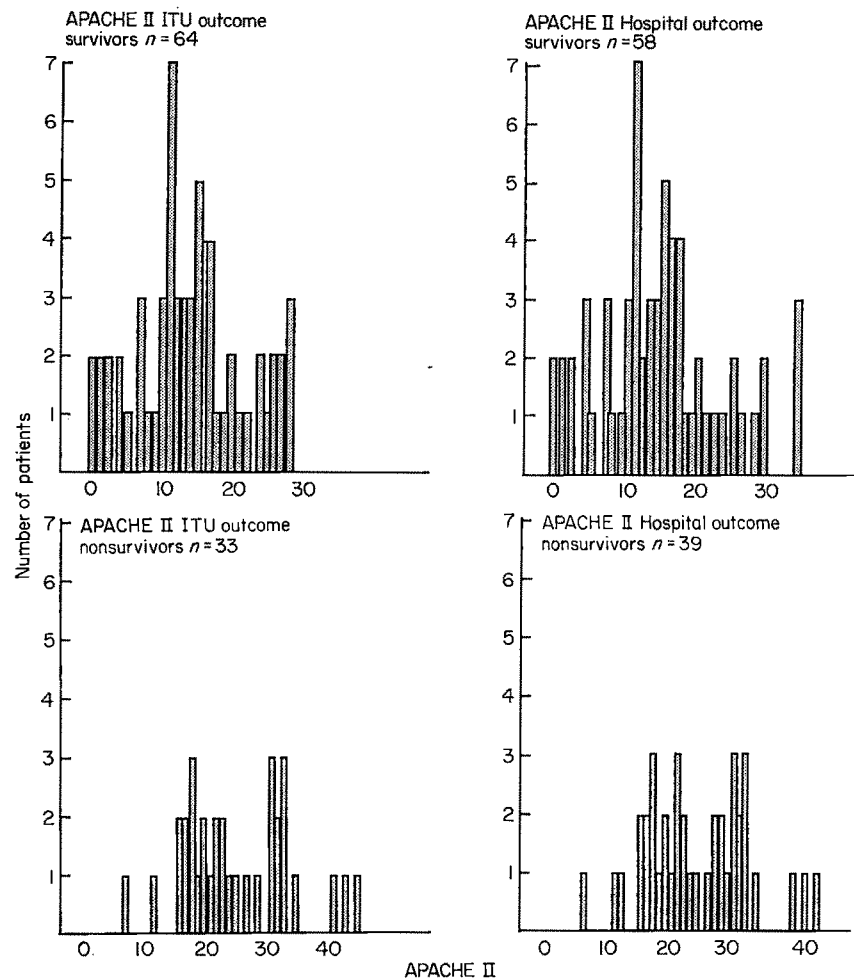


Fig. 2. APACHE II scores in survivors and nonsurvivors in respect of ITU and hospital outcome.

and 20 were taken as predictors of death ('test positive') for APACHE II, and scores greater or equal to 13 and 16 as 'test positive' for CSS because these were found to offer the optimal values in terms of both sensitivity and specificity.

A positive predictive value was calculated for each group for CSS and APACHE II using the following formula:

$$\text{Positive predictive value} = \frac{\text{true positive}}{\text{true positive} + \text{false positive}}$$

Rank Spearman correlation analysis was performed between APACHE II and CSS on the data from all 97 patients, from hospital survivors only, and from hospital nonsurvivors only, using the percentage of maximum score (because the maximum score was numerically different in each system).

### Results

Table 1 shows the characteristics of the 97 patients studied. Sixty-four of these patients survived ITU, and 58 survived to leave hospital. This is equivalent to an ITU mortality of 34.0% and a hospital mortality of 40.2%. One of the 59 survivors had died at home at the time of data collection (4 months after the end of the study).

The eight histograms (Figs 1 and 2) show the relationships between CSS or APACHE II scores and ITU or hospital outcome. All eight histograms refer to the same 97 patients. Survivors and nonsurvivors were compared in groups using Mann-Whitney *U* analysis and these groups and results are shown in Table 2. Median scores and ranges are given for each of the groups. *Z* is the normal approximation for the distribution of *U* and the *p* value is based on

Table 3. Sensitivity, specificity and positive predictive value of APACHE II and CSS for ITU and Hospital outcome. Death = 'Outcome positive'.

Group	Test positive	Sensitivity	Specificity	Positive predictive value
APACHE II ITU outcome	Score $\geq 18$	71%	73%	58%
APACHE II hospital outcome	Score $\geq 18$	75%	74%	66%
APACHE II ITU outcome	Score $\geq 20$	74%	64%	57%
APACHE II hospital outcome	Score $\geq 20$	80%	67%	67%
CSS ITU outcome	Score $\geq 13$	60%	85%	53%
CSS hospital outcome	Score $\geq 13$	61%	77%	58%
CSS ITU outcome	Score $\geq 16$	67%	71%	56%
CSS hospital outcome	Score $\geq 16$	64%	63%	56%

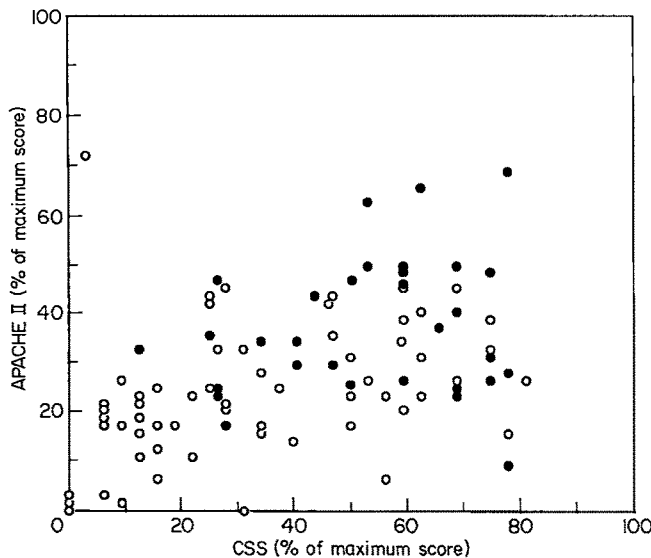


Fig. 3. Rank Spearman correlation between APACHE II and CSS. For survivors (○):  $r^2 = 0.37$ , for survivors+nonsurvivors:  $r^2 = 0.28$ , for nonsurvivors (●):  $r^2 = 0.0274$

the Z value. It is highly probable that survivors will score lower than nonsurvivors ( $p < 0.001$ ) using either APACHE II or CSS systems for ITU and hospital outcome. In either system, however, a high score can be associated with survival.

The sensitivity and specificity of each scoring system in predicting ITU and hospital outcome are shown in Table 3. APACHE II had a slightly better positive predictive value for both ITU and hospital outcome. Both systems were better predictors of hospital outcome than ITU outcome.

Figure 3 shows the correlation between APACHE II and CSS for all 97 patients, for the hospital survivors only and for the hospital nonsurvivors only. These are plotted as percentage of maximum score. The Rank Spearman coefficients for each group are shown in Table 4 and illustrate a good correlation for hospital survivors and nonsurvivors together ( $n = 97$ ), and for hospital survivors alone ( $n = 58$ ). Correlation for hospital nonsurvivors alone was poor ( $n = 39$ ).

### Discussion

At present, illness severity scoring systems do not predict outcome with 100% accuracy in ITU patients, and their use as prognostic indicators is of questionable value. For example, there are always a number of high-scoring survivors and low-scoring nonsurvivors, precluding a patient score from influencing the decision to withdraw or withhold therapy. However, scoring systems play a vital role in the audit of ITU performance and in clinical research.

The APACHE II system is used commonly to stratify

patient populations in clinical trials, and has rightly become the standard for this type of work. It may be less useful as an audit tool outside the teaching centres where a system must be simple and quick to complete if accuracy and compliance are to be achieved. Rigorous application of each component of the APACHE II score is essential to avoid error and bias; this is time-consuming and diverts ITU doctors from the patient's care. CSS might be an appropriate audit tool in ITUs where time and manpower are both limited.

In this study, CSS was not weighted for age and the score does not include weighting for diagnostic group or chronic health. In addition the physiological score is much simpler than that of APACHE II and can be completed within 2 minutes, by nursing staff if necessary. This study has shown that the information gained from CSS admission score is only marginally less powerful than that gained from APACHE II in terms of prediction of outcome, even though the amount of patient data required in completing CSS is very much less. The addition of a weighting for age and simple chronic health evaluation might improve CSS without destroying its simplicity, which is its fundamental strength.

The correlation between APACHE II and CSS was good for all 97 patients and slightly better for hospital survivors. It is interesting that the correlation for hospital nonsurvivors was poor. Most of the 39 hospital nonsurvivors attracted a high score in one or other system. It is possible that the poor correlation in the nonsurvivor group reflects a difference between high and low APACHE II and CSS scores. The scatter of scores is greater for high scores (which tend to be from nonsurvivors) than for low scores (which tend to be from survivors).

This study has highlighted the modest predictive power of an APACHE II admission score, which is almost matched by a CSS admission score. The significance of trends in APACHE II scores taken daily has been identified<sup>4</sup> and recent work suggests that a score after 48 hours of ITU treatment provides a better prediction of outcome than a score taken on admission (Winter RJ, personal communication). The results of the series of African patients scored with CSS<sup>6</sup> showed that a change in score between admission and day four was a significant indicator of outcome. Further work may identify the best time to record a single clinical sickness score and evaluate the power of serial score trends, producing simple dynamic models which could be used to influence decision-making in ITU.

### Acknowledgments

The authors thank the following for their advice during the preparation of this paper: Dr A. Hughes, University of Bristol, Department of Epidemiology, Professor C.

Table 4. Spearman rank correlation between APACHE II and CSS scoring systems.

Hospital outcome	<i>r</i>	<i>t</i>	d.f.	<i>p</i>
Survivors and nonsurvivors	0.5418 (0.5409)	6.2836	95	< 0.0001
Survivors	0.6102 (0.6089)	5.7640	56	< 0.0001
Nonsurvivors	0.1657 (0.1629)	1.0220	37	0.3134

*r* = Spearman rank correlation coefficient. Figures in brackets corrected for ties. d.f. = degrees of freedom.



Prys-Roberts and Dr S. M. Willatts, Sir Humphry Davy  
Department of Anaesthesia, Bristol.

### References

1. KNAUS WA, DRAPER EA, WAGNER DP, ZIMMERMAN JE. APACHE II: A severity of disease classification system. *Critical Care Medicine* 1985; **13**: 818-29.
2. JACOBS S, CHANG RWS, LEE B. One year's experience with the APACHE II severity of disease classification system in a general intensive care unit. *Anaesthesia* 1987; **42**: 738-44.
3. JACOBS S, CHANG RWS, LEE B. Audit of intensive care: a 30 month experience using the APACHE II severity of disease classification system. *Intensive Care Medicine* 1988; **14**: 567-74.
4. CHANG RWS, JACOBS S, LEE B, PACE N. Predicting deaths among intensive care unit patients. *Critical Care Medicine* 1988; **16**: 34-42.
5. TURNER JS, POTGIETER PD, LINTON DM. Systems for scoring severity of illness in intensive care. *South African Medical Journal* 1989; **76**: 17-20.
6. WATTERS DAK, WILSON IH, SINCLAIR JR, NGANDU N. A clinical sickness score for the critically ill in Central Africa. *Intensive Care Medicine* 1989; **15**: 467-70.

### Appendix 1 Clinical Sickness Score.

	Scores								
	4	3	2	1	0	1	2	3	4
Heart rate (beats/minute)	> 180	140–179	110–139		70–109		55–69	40–54	< 40
Systolic blood pressure (mmHg)	> 200	170–199	150–169	140–149	100–139	80–99	60–79		< 60
Respiratory rate (/minute)	> 50	35–49		25–34	12–24	10–11	6–9		IPPV
Urine output (ml/hour)	anuria		< 50		50–200		> 200		
Temperature (°C)	≥ 40	39		38	36–37	33–35	31–32	30	20
Glasgow coma scale	subtract from 15								
Age (years)	61 +		51–60	41–50	< 40				

From Watters et al.<sup>6</sup>

## Comparison of extradural and intravenous diamorphine as a supplement to extradural bupivacaine

A. LEE, D. McKEOWN, M. BROCKWAY, J. BANNISTER AND J. A. W. WILDSMITH

### Summary

*The influence of route of administration (extradural as compared with intravenous) of diamorphine 0.5 mg/hour as a supplement to extradural bupivacaine (0.125% at 15 ml/hour) was investigated in two groups of 20 patients who underwent major abdominal gynaecological surgery. Significantly more patients in the intravenous group withdrew because of inadequate analgesia ( $p < 0.05$ ). Those in the extradural group were significantly more drowsy throughout the study ( $p < 0.01$ ), but no major side effects were encountered.*

### Key words

*Anaesthetic techniques, regional; extradural.*

*Anaesthetics, local; bupivacaine.*

*Analgesics; diamorphine.*

*Pain; postoperative.*

Continuous extradural infusions of local anaesthetic drugs are being used with increasing frequency for postoperative analgesia,<sup>1–3</sup> but it is well recognised that total pain relief does not always ensue.<sup>4</sup> Pain fibres may traverse non-somatic nerves and other sources of discomfort assume greater importance once adequate wound analgesia is achieved. As a result as many as 50% of patients require further medication, usually in the form of an opioid.<sup>5</sup> Combined extradural administration of a local anaesthetic and an opioid has been shown to be more consistently effective than either drug given alone after various types of surgery.<sup>2,5,6</sup> However, it has not been clearly established that there is an advantage in giving the opioid extradurally, as opposed to systemically, in the presence of an effective local anaesthetic block. A small dose of systemic opioid might be sufficient to supplement the extradural analgesia and obtund discomfort from outside its area of effectiveness without the risk of delayed respiratory depression. This study was designed to compare the clinical effectiveness of the administration of diamorphine by either the extradural or intravenous route in the presence of a continuous postoperative extradural local anaesthetic block.

### Methods

Forty patients (ASA 1 or 2, aged 18–70 years), took part in the study which was approved by the area ethics commit-

tee. All patients underwent major gynaecological surgery through a lower abdominal incision and received a standard anaesthetic technique. Before surgery, and after receiving a full explanation, the patients completed a series of visual analogue scales and a symptom checklist identical to those used in the postoperative assessments. It was clearly explained to the patients that should they consider their postoperative pain relief inadequate, the study infusions would be discontinued and morphine given intramuscularly at their request.

Diamorphine 5 mg was given intramuscularly one hour before surgery. An intravenous infusion of compound sodium lactate solution was started in the anaesthetic room after which an extradural catheter was sited at the 10th or 11th thoracic interspace. Four ml of 2% plain lignocaine was administered as a test dose and if this produced no obvious effect a further 16 ml of 2% plain lignocaine was given over not less than 2 minutes. General anaesthesia was induced with thiopentone and maintained with nitrous oxide 70% and enflurane 1% in oxygen. The patients breathed spontaneously from a facemask. Ten ml of bupivacaine 0.5% plain solution was given extradurally after one hour or at wound closure, whichever occurred first. The patients were then randomly divided into two groups for postoperative analgesia.

All patients received an extradural infusion after surgery, of 0.125% bupivacaine hydrochloride at 15 ml/hour. In

A. Lee, FCAnaes, M. Brockway, FCAnaes, J. Bannister,\* FCAnaes, Senior Registrars, D. McKeown, FCAnaes, J.A.W. Wildsmith, MD, FCAnaes, Consultants, Department of Anaesthesia, Royal Infirmary, Edinburgh EH3 9YW.

\*Present address: Department of Anaesthesia, Ninewells Hospital, Dundee.

Accepted 3 September 1990.

**Table 1.** Details of patients (SD).

	Intravenous	Extradural
Age; years	44 (7.7)	41.7 (9.3)
Height; m	1.60 (0.05)	1.62 (0.08)
Weight; kg	64.3 (8.4)	67 (11.6)
Extradural site		
T <sub>10-11</sub>	16	18
T <sub>11-12</sub>	4	2
Incision		
Transverse	18	17
Midline	2	3
Duration of operation; minutes	91.4 (35)	80.3 (28)
Blood loss; ml	270 (442)	280 (489)

addition every patient received 0.5 mg/hour of diamorphine hydrochloride. In one group the diamorphine was mixed with the local anaesthetic solution and administered extradurally. These patients received 0.9% saline 2 ml/hour intravenously. In the other group the diamorphine was given intravenously (0.25 mg/ml diamorphine in 0.9% saline at 2 ml/hour). These patients received only local anaesthetic solution extradurally. An Imed 960 pump was used to administer the extradural solution. A 30-ml paediatric burette situated between the bag of solution and the pump was refilled at 2-hourly intervals by nursing staff. The intravenous solution was given through a dedicated intravenous cannula using a Vickers Treonic syringe driver. Both infusions were prepared by the hospital pharmacy and started immediately after surgery. Medical staff were not aware of the contents of either the intravenous or the extradural solution.

The upper dermatomal level of the extradural block to pinprick was determined as soon as the patient was awake enough to cooperate. Motor power in the legs was graded at the same time using a 5 point scale (0 = full power; 1 = weak, but able to raise against gravity; 2 = good movement, gravity compensated; 3 = minimal movement; 4 = paralysis). Assessments at 2, 4, 6, 12 and 21 hours after operation included completion of a patient symptom checklist and 10-cm visual analogue scales (no pain—the worst pain I can imagine; alert—drowsy; steady—dizzy; muzzy—clearheaded; clumsy—well coordinated; very well—very ill). Block height and motor power were assessed as before and an overall observer rating made by the anaesthetist using a 10-cm visual analogue scale (extremely poor—extremely good).

Heart rate and blood pressure were recorded at regular, frequent intervals by nursing staff, as were hourly respiratory rates and volumes of solution infused extradurally and intravenously. Such nursing observations were routine and distinct from the formal assessments made for the study.

Comparisons between groups were made using Mann-Whitney tests with Bonferroni corrections, Chi-squared or Fisher's exact tests as appropriate.

### Results

The groups were comparable in age, height, weight, extradural catheter site, type of incision (transverse or midline), duration of operation and blood loss (Table 1). There were no significant differences between the groups in sensory or motor block immediately after the operation (Table 2). One

**Table 2.** Analgesic levels to pinprick and degree of motor blockade immediately after operation (SD). Most dense block recorded when motor blockade differed between legs.

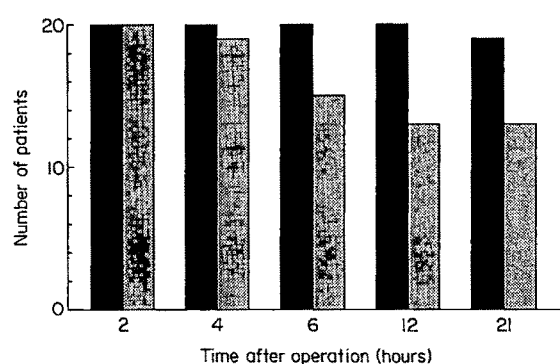
	Intravenous	Extradural
Analgesic level		
Median	T <sub>4</sub>	T <sub>4</sub>
Quartiles	T <sub>3</sub> -T <sub>6</sub>	T <sub>3</sub> -T <sub>5</sub>
Mean (SD)	T <sub>4.3</sub> (1.7)	T <sub>4</sub> (1.7)
Motor blockade		
0	0	0
1	2	2
2	3	4
3	7	7
4	8	7

patient in the extradural group was withdrawn between 12 and 21 hours because of a technical problem with the extradural infusion pump. One patient in the intravenous group was withdrawn between 6 and 12 hours because of slight numbness and heaviness in one arm; pinprick testing demonstrated a block to C<sub>8</sub> on that side. There were no problems associated with this and no treatment required, but it was thought unwise to continue the infusion at the predetermined rate. Six other withdrawals were because analgesia was considered inadequate by the patient and all six were in the intravenous group ( $p < 0.05$ ) (Fig. 1).

When the visual analogue scores of those patients who had not been excluded because of inadequate pain relief were compared, there was, as would be expected, no significant difference between the groups.

Patients receiving diamorphine extradurally were significantly less alert from 4 hours onwards ( $p < 0.01$ ) and still judged themselves to be markedly drowsy the morning after surgery. In contrast, patients receiving intravenous diamorphine became significantly more alert during the study period. There were no other significant differences between the groups in visual analogue scores, but patients receiving extradural diamorphine consistently judged themselves to be less well coordinated and less clear headed than patients receiving intravenous diamorphine from 6 hours onwards (range  $p < 0.013$ – $p < 0.041$ ).  $p$  values of  $< 0.01$  were considered significant to allow for the performance of multiple tests.

The upper level of analgesia to pinprick regressed in both groups during the first 4 hours, but was well maintained subsequently (Fig. 2). There were no significant differences

**Fig. 1.** Number of patients remaining in the study at each time period. ■, extradural group; ▨, intravenous group.

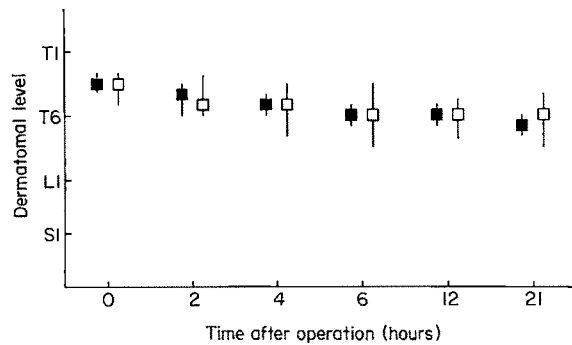


Fig. 2. Upper level of block to pinprick. ■, extradural (median quartile); □, intravenous (median quartile).

between the groups in block height at any time. One patient in the extradural group and four in the intravenous group developed unilateral blockade. Two of the patients in the intravenous group with unilateral blocks withdrew from the study with higher pain scores after requesting additional analgesia. The difference in the incidence of unilateral block between the groups was not significant.

Motor block regressed rapidly in the majority of patients, and the degree of motor blockade in patients remaining in the study at 21 hours is shown in Table 3. There were no differences between the groups at any time.

Side effects are recorded in Table 4; the only significant difference between groups was the increased incidence of pruritus in patients receiving extradural diamorphine ( $p < 0.01$ ). Treatment was not required for this in any patient. No patient developed respiratory depression defined as a respiratory rate of less than 10 breaths/minute. Respiratory rate was counted over one minute at hourly intervals. No patient required treatment for hypotension. Patients were routinely catheterised for 24 hours by the surgeons and it was not possible to determine if there was a difference in the incidence of urinary retention between the techniques.

### Discussion

We have previously shown that the combination of extradural bupivacaine and diamorphine is more effective for postoperative analgesia than either drug given alone.<sup>5</sup> This study has shown that diamorphine is more effective as a supplement to extradural bupivacaine when administered by the extradural rather than the intravenous route. Extradural infusion of a low dose of an opioid alone may provide sufficient pain relief after abdominal surgery and may be more effective than intravenous administration.<sup>7,8</sup> Profound analgesia can be obtained even when systemic drug levels are low or undetectable.<sup>9</sup> The improved pain relief obtained when opioids are given by the extradural

Table 3. Degree of motor blockade at 21 hours. Most dense block recorded when motor blockade differed between legs.

Degree of motor blockade	Intravenous	Extradural
0	5	11
1	3	6
2	2	1
3	2	1
4	1	0

Table 4. Number of patients with side effects. A single recording at any time during the study is taken as a positive finding.

	Intravenous	Extradural
Respiratory depression; < 10 breaths/minute	0	0
Hypotension requiring treatment	0	0
Pruritus	5	16
Nausea and/or vomiting	11	12
Shoulder pain	5	6

route appears to be maintained and clinically relevant even in the presence of an effective local anaesthetic block.

These findings help to confirm that the action of extradural diamorphine is from direct diffusion to the central nervous system at the spinal cord level rather than by absorption into the circulation and subsequent delivery to the brain. It would seem likely that the diamorphine acts at the spinal level, but it must be noted that extradural diamorphine produced a greater degree of sedation. This could have been secondary to improved analgesia, but there is also the possibility that the extradural route of administration has a greater supraspinal action. If this is the case it would be from cephalad spread of diamorphine within the cerebrospinal fluid as has been demonstrated with fentanyl.<sup>10</sup> The combined extradural administration of these drugs will raise concern about the risk of complications. Cardiovascular variables remain very stable during postoperative extradural infusion of bupivacaine and are unaffected by the addition of an opioid. Respiratory depression remains the major concern and has been reported after the extradural use of most lipophilic opioids as well as the more water soluble morphine.<sup>11-13</sup> It probably occurs secondary to cephalad migration of drug in the cerebrospinal fluid and may take several hours to develop with morphine because of slow transfer across the dura. Respiratory depression occurs more rapidly after lipophilic agents,<sup>14</sup> although clearance from the cerebrospinal fluid is much more rapid than morphine. It is likely that respiratory depression after extradural administration of a lipophilic opioid depends on the administration of a large bolus dose, allowing sufficient drug to reach the brainstem.<sup>15</sup> We administer extradural opioids by infusion only, in the hope of reducing this possibility. A lipophilic drug should be removed from the cerebrospinal fluid at a relatively fast rate,<sup>15</sup> and it is suggested that avoidance of bolus administration will prevent cephalad spread. However, many more patients will need to be studied to confirm this hypothesis.

Urinary retention is a common side effect of combined extradural bupivacaine and diamorphine infusion and may not be alleviated by intravenous naloxone administration. Urinary catheterisation may be perfectly acceptable after some surgical procedures, but the possibility should be considered in advance.<sup>13</sup> Pruritus is more frequent when opioids are given by the extradural route, but is usually of a minor nature and seldom requires treatment. It is readily relieved with intravenous naloxone.<sup>5</sup> The incidence of nausea and vomiting associated with the use of extradural opioids is similar to the incidence after systemic administration.<sup>8</sup>

We conclude that the extradural administration of diamorphine is more effective as a supplement to extradural bupivacaine than intravenous diamorphine, but that

the patients are drowsier. A fixed dose of diamorphine was used to allow direct comparison of route of administration. More flexibility in the dose of opioid (i.e. by patient controlled analgesia) might have produced equally effective supplementation to the extradural bupivacaine, but of course involves greater complexity. Further, the doses of bupivacaine and diamorphine used here are reasonably effective on their own,<sup>5</sup> and the degree of sedation and motor block may be decreased by using a combination at lower dose levels. Both these aspects require to be investigated.

### References

1. FISCHER RL, LUBENOW TR, LICEAGA A, MCCARTHY RJ, IVANKOVICH AD. Comparison of continuous epidural infusion of fentanyl-bupivacaine and morphine-bupivacaine in management of postoperative pain. *Anesthesia and Analgesia* 1988; **67**: 559–63.
2. CULLEN ML, STAREN ED, EL-GANZOURI A, LOGAS WG, IVANKOVICH AD, ECONOMOU SG. Continuous epidural infusion for analgesia after major abdominal operations: a randomized, prospective, double-blind study. *Surgery* 1985; **98**: 718–28.
3. RAJ PP, KNARR DC, VIGDORTH E, DENSON DD, PITHER CE, HARTRICK CT, HOPSON CN, EDSTROM HH. Comparison of continuous epidural infusion of a local anesthetic and administration of systemic narcotics in the management of pain after total knee replacement surgery. *Anesthesia and Analgesia* 1987; **66**: 401–6.
4. CONACHER ID, PAES ML, JACOBSON L, PHILLIPS PD, HEAVISIDE DW. Epidural analgesia following thoracic surgery. A review of two years' experience. *Anaesthesia* 1983; **38**: 546–51.
5. LEE A, SIMPSON D, WHITFIELD A, SCOTT DB. Postoperative analgesia by continuous extradural infusion of bupivacaine and diamorphine. *British Journal of Anaesthesia* 1988; **60**: 845–50.
6. LOGAS WG, EL-BAZ N, EL-GANZOURI A, CULLEN M, STAREN E, FABER LP, IVANKOVICH AD. Continuous thoracic epidural analgesia for postoperative pain relief following thoracotomy: a randomized prospective study. *Anesthesiology* 1987; **67**: 787–91.
7. EISENACH JC, GRICE SC, DEWAN DM. Patient-controlled analgesia following cesarean section: a comparison with epidural and intramuscular narcotics. *Anesthesiology* 1988; **68**: 444–8.
8. HARRISON DM, SINATRA R, MORGESE L, CHUNG JH. Epidural narcotic and patient-controlled analgesia for post-cesarean section pain relief. *Anesthesiology* 1988; **68**: 454–7.
9. MORGAN M. The rational use of intrathecal and extradural opioids. *British Journal of Anaesthesia* 1989; **61**: 165–88.
10. GOURLAY GK, MURPHY TM, PLUMMER JL, KOWALSKI SR, CHERRY DA, COUSINS MJ. Pharmacokinetics of fentanyl in lumbar and cervical CSF following lumbar epidural and intravenous administration. *Pain* 1989; **38**: 253–9.
11. BROCKWAY MS, NOBLE DW, SHARWOOD-SMITH GH, MCCLURE JH. Profound respiratory depression after extradural fentanyl. *British Journal of Anaesthesia* 1990; **64**: 243–5.
12. WATSON J, MOORE A, MCQUAY H, TEDDY P, BALDWIN D, ALLEN M, BULLINGHAM R. Plasma morphine concentrations and analgesic effects of lumbar extradural morphine and heroin. *Anesthesia and Analgesia* 1984; **63**: 629–34.
13. READY LB, ODEN R, CHADWICK HS, BENEDETTI C, ROOK GA, CAPLAN R, WILD LM. Development of an anesthesiology-based postoperative pain management service. *Anesthesiology* 1988; **68**: 100–6.
14. GOURLAY GK, CHERRY DA, PLUMMER JL, ARMSTRONG PJ, COUSINS MJ. The influence of drug polarity on the absorption of opioid drugs into CSF and subsequent cephalad migration following lumbar epidural administration: application to morphine and pethidine. *Pain* 1987; **31**: 297–305.
15. SJÖSTRÖM S, HARTVIG P, PERSSON P, TAMSEN A. Pharmacokinetics of epidural morphine and meperidine in humans. *Anesthesiology* 1987; **67**: 877–88.

## Propofol in short gynaecological procedures

Comparison of recovery over 2 days after anaesthesia with propofol or thiopentone as sole anaesthetic agent

L. D. SANDERS, P. A. CLYBURN, M. ROSEN AND J. O. ROBINSON

### Summary

Recovery was assessed over 48 hours after anaesthesia with propofol or thiopentone as sole anaesthetic agent in 36 unpremedicated gynaecological patients. Immediate recovery, as measured by the Steward scale, was shown to be quicker for the patients given propofol. At one hour postoperatively the thiopentone group showed impaired visual-motor coordination on the aiming test ( $p < 0.01$ ) and dexterity task ( $p < 0.05$ ), and a slowing of reaction time ( $p < 0.01$ ). Patients given propofol showed only an increase in reaction time ( $p < 0.05$ ). By 2 hours the thiopentone group showed impairment only in the aiming task ( $p < 0.05$ ). No further significant impairment was detected at 4, 24 or 48 hours. However, patients reported symptoms throughout the 48 hours indicative of residual drug effects. There was a substantial practice effect with some tests which may have obscured impairment. It can be argued therefore that the better recovery profile after propofol is still evident at 24 hours.

### Key words

Anaesthetics, intravenous; thiopentone, propofol.

Anaesthesia; recovery.

Psychomotor, tests.

'Diprivan' (propofol) is an intravenous induction agent formulated in an aqueous soya bean oil emulsion and is rapidly redistributed and metabolised, making it suitable for maintenance of anaesthesia.<sup>1,2</sup> Initial recovery is rapid, as assessed by time to eye-opening,<sup>3</sup> ability to give correct date of birth,<sup>4</sup> and reaction time.<sup>5</sup> Such assessments give a crude guide to the speed of initial recovery, but may not be relevant to the decision to discharge a patient. Of greater importance, particularly with short-stay patients, is the time taken to return completely to the pre-anaesthetic state.

This study assesses the quality of anaesthesia and psychomotor recovery of patients over 2 days following anaesthesia with either propofol or thiopentone, used as sole anaesthetic agents for short gynaecological procedures.

### Method

The study was approved by the Hospital Ethics Committee. ASA 1 and 2 patients, aged between 16 and 65 years, scheduled for minor gynaecological procedures after giving informed consent, were randomly allocated to two groups, which received either propofol or thiopentone as sole anaesthetic agent. Exclusions were made in the case of

previous adverse experience of general anaesthesia, pregnancy, gross obesity, respiratory, cardiovascular, hepatic, renal, haemopoietic or endocrine impairment and those taking any drugs or medication likely to influence the course of anaesthesia.

### Procedure

Both the patient and the psychologist who assessed recovery were blind to the identity of the anaesthetic agent. During the pre-operative visit the patient was instructed and allowed to become familiar with the psychometric tests, after which baseline measurements were taken.

Patients were unpremedicated. On arrival in the operating theatre an 18-gauge cannula was inserted in a peripheral vein and flushed with 0.9% saline. Baseline pulse and blood pressure readings were taken using a Hewlett-Packard HP 78352A monitor. Anaesthesia was induced with an initial bolus of either propofol (2.5 mg/kg) or thiopentone (4 mg/kg) injected over 30 seconds. Repeat boluses of either propofol (20–40 mg) or thiopentone (50–100 mg) were administered to maintain adequate anaesthesia, as indicated by response to surgical stimuli.

L.D. Sanders, BScEcon, PhD, AFBPsS, C Psychol, Research Fellow, P.A. Clyburn, MB, BS, MRCP, FCAnaes, Senior Registrar, M. Rosen, MB, ChB, FCAnaes, Consultant and Honorary Professor, Department of Anaesthesia, University of Wales, College of Medicine, Heath Park, Cardiff CF4 4XW, J.O. Robinson, BSc, PhD, FBPSS, C Psychol, Senior Lecturer, School of Psychology, University College of Cardiff, PO Box 78, Cardiff, CF1 1XL.

Accepted 17 October 1990.



The patient breathed 100% oxygen delivered by a coaxial system (Penlon). Blood pressure and pulse rate were recorded 2 minutes after induction and subsequently at 5-minute intervals. The occurrence of apnoeic episodes, coughing, twitching, hiccups or movement during induction or maintenance was recorded, and the control of the depth of anaesthesia was graded as 'good', 'moderate' or 'poor'.

In the recovery room the times at which each patient was able to open the eyes, protrude the tongue, give a correct date of birth, sit up and stand unaided were recorded. Assessments of breathing, movement and the degree of wakefulness were made on the Steward Scale<sup>6</sup> at 3, 5, 15 and 30 minutes after the last bolus, and on discharge from the recovery ward.

Psychomotor performance was assessed at 1, 2, 4, 24 and 48 hours after the end of anaesthesia.

#### Psychometric tests

The tests in the battery assessed cognitive functioning, visual-motor coordination, reaction time and ataxia. All six of the tests in the battery were standard psychometric measures with established use in psychological experimentation.

**Stroop test.** Two sets of 25 cards, on each of which is printed one word in a coloured ink, were presented to the patient who was requested to state the colour of the inks. On one set, the nonconflict set, the words were animal names, whilst on the conflict set the names of colours were printed; the names were different from the colour used for that word. The speed of reading each card set was recorded; the difference between the two times was an indication of ability to concentrate or attend to the task in hand.<sup>7</sup> The Stroop test is considered to examine impairment of high level intellectual function, and has been shown to be an effective measurement in psychopharmacology.<sup>8-10</sup>

**Digit span.** Strings of numbers increasing in length from three upwards were recited to the subject who had to repeat the string correctly, but in reverse order. This was continued until two consecutive errors were made; this gives an indication of short-term auditory memory capacity.<sup>11</sup>

**Aiming.** Three hundred 2-mm diameter circles linked in lines of 20 across a sheet of paper were presented to the subject who was asked to place a dot inside each circle within a time limit of 90 seconds. The number of dots correctly placed within the circles are recorded. This assesses hand-eye coordination;<sup>12</sup> both visual and motor processes have to function together.

**Dexterity.** The task is to guide a loop along a twisted length of wire; when the loop touches the wire a buzzing sound is emitted. For each hand the time taken was recorded as well as the number of touches; the score was the mean of the scores for the two hands. Although this task incorporates a visual component, it primarily assesses manual dexterity.

**Choice reaction time.** This was measured by an apparatus with six buttons, arranged in an arc against a black background at equal distances from a central point, which light up in a randomised order. The task was to press the buttons as soon as the light appeared each time returning the finger to a central point; the time taken to do this was recorded by the apparatus.

**Ataxia.** The subject stood attached to an ataxiometer which measures the amount of forwards and backwards sway during a period of 15 seconds whilst the patient stands, unsupported, with closed eyes.<sup>13</sup>

#### Subjective effects

At 4, 24 and 48-hour follow-up, patients were presented with a checklist of the following subjective after effects: shivering, feeling hot, sleepiness, headache, cough, nausea, vomiting, clumsiness, confusion, double vision, flush, depression, elation and weakness.

The psychometric data were tested using a paired *t*-test or a Wilcoxon matched pairs test to seek evidence of impairment within each group, and an analysis of covariance technique, (ANOVA) or a Mann-Whitney test, as appropriate to assess the differential drug effect. A Chi-squared test was applied to the categorical variables.

#### Results

Data were collected for 36 patients; 18 received thiopentone and 18 propofol.

The details of the patients, as given in Table 1, show the groups to be comparable in weight and height, but by chance the mean age of the propofol group was greater than that of the thiopentone group. The mean induction dose of propofol was 158 mg, and of thiopentone was 267 mg; the mean maintenance dose was 123 mg and 501 mg respectively. Thus the mean total doses were 284 mg of propofol and 800 mg of thiopentone; this produced a mean mg/kg dose of 4.43 (SD 0.45) and 12.06 (SD 3.75), respectively.

The quality of induction and maintenance of anaesthesia was satisfactory in all patients. There was no significant difference between the groups in either the duration of surgery or of anaesthesia (Table 2). In the propofol group there were four patients for whom instances of moderate movement in response to surgery were recorded, and 11 of those given thiopentone; this difference was significant ( $p < 0.05$ ). The control of depth of anaesthesia was recorded as 'good' in 10 cases with propofol and in two cases with thiopentone ( $p < 0.05$ ). At induction there were eight recorded side effects (twitching or hiccups) after

Table 1. Details of patients, mean (SD).

	Propofol <i>n</i> = 18	Thiopentone <i>n</i> = 18
Age; years	47.6 (10.4)	38.2 (8.7)
Weight; kg	63.6 (7.3)	64.5 (8.6)
Height; cm	159.4 (8.2)	162.4 (6.6)
Dose (mg)		
Induction	158.3 (21.5)	267.5 (47.7)
Maintenance	123.1 (31.8)	501.4 (236.0)
Overall mg/kg dose	4.4 (0.5)	12.1 (3.8)

**Table 2.** Duration of surgery and recovery times in minutes, mean (SD).

	Propofol	Thiopentone
Duration of surgery	7.2 (3.3)	6.4 (4.4)
First to last dose	9.9 (4.2)	9.7 (4.6)
End of surgery to: opening eyes†	6.7 (3.4)	21.4 (9.1)
protruding tongue‡	7.3 (3.4)	22.1 (9.4)
orientation‡	8.7 (3.4)	23.2 (9.4)
sitting up*	43.0 (20.2)	77.8 (57.1)
standing up*	80.0 (39.4)	115.2 (53.6)

Significant difference between groups: \* $p < 0.05$  † $p < 0.001$ .

propofol and two after thiopentone, whilst during maintenance there were two and six respectively. Apnoeic episodes were recorded for nine patients given propofol and eight given thiopentone. The recovery times for the propofol group were significantly shorter than those for the thiopentone group throughout their stay in the recovery room (Table 2). Furthermore, the data from the Steward Scale showed that the propofol group had significantly higher scores at each point (Table 3).

#### Psychometric Results

Two patients were in too much discomfort to complete the first postoperative test, and one was too nauseated to do the first two postoperative sessions; all three had received thiopentone.

There was no significant evidence of impairment in either group at any point with the cognitive tests, but there was a significant improvement in the Stroop time at 2 hours and in the digit span at 48 hours ( $p < 0.05$ ).

**Table 3.** The Steward Scores, mean (SD).

	Propofol	Thiopentone
3 minutes*	2.9 (2.2)	1.2 (1.3)
5 minutes‡	4.9 (1.8)	1.0 (0.5)
15 minutes‡	5.9 (0.2)	2.1 (1.8)
30 minutes†	5.9 (0.2)	4.6 (1.9)
At discharge to ward†	6.0 (0.0)	5.6 (0.5)

Significant difference between groups: \* $p < 0.05$ , † $p < 0.01$ , ‡ $p < 0.001$ .

There was significant impairment in visual-motor co-ordination for the thiopentone group, as shown by the increase in errors in the dexterity task at 1 hour, and the difference between groups was shown to be significant ( $p < 0.05$ ). Furthermore, patients given propofol completed the task more quickly than at baseline, whilst the time taken by those given thiopentone had increased; the effect of the drug was a significant factor at this point ( $p < 0.05$ ). There was also a significant decrease in the score of the thiopentone group in the aiming task at 1 and 2 hours ( $p < 0.001$  and  $p < 0.05$ ), and again the drug group was shown to be a factor ( $p < 0.001$  and  $p < 0.05$ ). In this task there was significant improvement compared with baseline measurements in the performance from 4 hours onwards ( $p < 0.01$ ) for the patients given propofol, whereas the thiopentone group did not begin to show this until the next day ( $p < 0.01$ ). An analysis of variance showed the drug to be a significant factor at 48 hours ( $p < 0.05$ ).

For the motor tasks there was no evidence of significant impairment of balance, as measured by the ataxiometer, but five patients given propofol and 10 given thiopentone were too drowsy to attempt this task. There was an increase in reaction time for both groups at 1 hour ( $p < 0.05$ ), and

**Table 4.** Psychometric results by anaesthetic agent.

Test mean (SD)	Baseline		1 hour		2 hours		4 hours		24 hours		48 hours	
	Pr	Th	Pr	Th	Pr	Th	Pr	Th	Pr	Th	Pr	Th
Stroop; seconds	5.3 (4.7)	5.5 (5.1)	3.5 (3.0)	5.1 (7.9)	*3.7 (4.7)	*2.8 (3.7)	4.7 (5.0)	4.3 (3.0)	5.6 (4.3)	3.5 (3.6)	5.3 (6.5)	3.9 (2.6)
Digit span	4.5 (0.9)	4.6 (1.1)	4.4 (1.0)	4.1 (1.1)	4.4 (0.9)	4.4 (1.0)	4.4 (0.9)	4.5 (0.8)	4.8 (1.3)	4.8 (1.0)	*5.1 (1.1)	*5.1 (0.8)
Aiming total	151 (39)	156 (30)	153 (40)	†118 (28)	161 (50)	*135 (35)	†172 (45)	152 (35)	†168 (40)	162 (34)	†180 (48)	†175 (41)
CRT; mseconds	73 (14)	74 (16)	*79 (19)	†93 (17)	75 (16)	82 (17)	72 (12)	77 (11)	71 (14)	73 (13)	*66 (13)	*68 (11)
Ataxia	20 (11)	18 (7)	19 (14)	27 (14)	23 (22)	28 (26)	16 (13)	20 (14)	15 (10)	17 (8)	*15 (9)	20 (11)
Dexterity time; seconds	23 (6)	24 (9)	21 (7)	28 (14)	19 (6)	26 (13)	20 (6)	26 (16)	23 (7)	27 (14)	22 (9)	25 (14)
Dexterity errors, median; test score-baseline score			-0.75	*6.5	-1.5	1.0	0.75	0.5	-1.0	0	-3.0	-0.5

Significant difference compared to baseline: \* $p < 0.05$ , † $p < 0.01$ , ‡ $p < 0.001$ .

Table 5. Symptoms check-list.

	4 hours		24 hours		48 hours	
	Pr	Th	Pr	Th	Pr	Th
Sleepiness	9	13	3	6	2	3
Headache	8	5	5	3	2	2
Weakness	5	6	4	3	4	4
Clumsiness	2	*8	1	3	—	1
Blurred vision	—	†7	1	1	3	0
Elation	2	5	—	1	1	1
Nausea	2	4	—	3	—	1
Confusion	1	4	—	1	2	1
Hot	4	1	4	3	4	1
Shivering	2	2	1	—	—	—
Flushed	1	2	1	1	1	1
Depressed	1	1	—	—	1	1
Vomiting	1	1	0	1	—	—

Significant difference between groups: \* $p < 0.05$ , † $p < 0.01$ .

the drug group was shown to be a factor ( $p < 0.05$ ). By the following day, however, there was an improvement compared with baseline measurements in the reaction times of both groups ( $p < 0.05$ ).

#### Subjective effects results

At 4 hours the most commonly reported symptoms were sleepiness, headache and weakness and there was no difference between groups. However, significantly more of the thiopentone group reported clumsiness and blurred vision. By 24 hours the incidence of these symptoms had reduced across the sample (Table 5).

#### Discussion

Although both drugs produced satisfactory anaesthesia it would seem that propofol provided a better quality. As patients given thiopentone responded to surgery more frequently, they were given a relatively larger total dose. Whilst the induction dose was within the range of equipotency,<sup>14-16</sup> the maintenance dose was not. However, as the dose given was that adequate to maintain anaesthesia for both groups of patients it is reasonable to conclude that these doses should also be considered equipotent when anaesthesia is to be maintained by infusion of these drugs as sole agents.

Immediate recovery was much quicker with propofol which accords with others studies.<sup>3,4</sup> The psychometric testing showed a better recovery profile for the patients given propofol than for those given thiopentone. Indeed it is only in their choice reaction times that impairment of test performance was evident, and this only at the one hour test point. In contrast, patients who received thiopentone showed impaired performance at one hour on the aiming, choice reaction time and dexterity tasks; this indicated a disruption in visual-motor coordination.

By 4 hours the performance of both groups in all tasks was not significantly poorer than their baseline performance. However, there is evidence of practice effects with the aiming task, digit span and choice reaction time as the performance of these tests had significantly improved from baseline by 48 hours for both groups. It has been shown

that improvement through learning may obscure persisting impairment.<sup>17,18</sup> In this study the group given thiopentone took longer to improve their performance in the aiming task, and this impediment to learning can be considered a form of cognitive impairment. Therefore it is necessary to admit the possibility of impairment in the thiopentone group up to 48 hours after the operation.

Performance on the Stroop test may also have been affected by practice effects since this was significantly better than baseline at 2 hours but not at 4 hours. This test point was only one hour after the previous session, and it is possible that this small time lag helped improve performance. The practice effects noted in these tests suggests they are of limited use in a patient population in which adequate pre-trial practice is rarely practicable.

The data from the symptom checklist indicate that whilst for most patients such symptoms may not persist into the first operative day, for a few they may still be present on the second day.

The results of this study indicate that when anaesthesia is maintained by an infusion of a single agent, propofol provides advantages over thiopentone in the quality of anaesthesia and of recovery. The subjective reporting of symptoms suggests that even after surgery of such brevity there may still be hangover effects for up to 48 hours. Although there is no evidence from the psychometric data of functional impairment at this stage, it is not possible to conclude that this represents full recovery. The possibility that the tests were not adequately sensitive cannot be excluded.

#### References

- GLEN JB, HUNTER SC. Pharmacology of an emulsion formulation of ICI 35-868. *British Journal of Anaesthesia*. 1984; **56**: 617-26.
- UPPINGTON J, KAY NH, SEAR JW. Propofol ('Diprivan') as a supplement to nitrous oxide-oxygen for the maintenance of anaesthesia. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 80-3.
- GRANT IS, MACKENZIE N. Recovery following propofol ('Diprivan') anaesthesia—a review of three different anaesthetic techniques. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 133-7.
- MILLAR JM, JEWKES CF. Recovery and morbidity after daycase anaesthesia. *Anaesthesia* 1988; **43**: 738-43.
- HERBERT M, MAKIN SW, BOURKE JB, HART EA. Recovery of mental abilities following general anaesthesia induced by propofol ('Diprivan') or thiopentone (Abstract). *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 132.
- STEWART DJ. A simplified scoring system for the post-operative recovery room. *Canadian Anaesthetists' Society Journal* 1975; **22**: 111-3.
- STROOP JR. Studies of interference in serial verbal reactions. *Journal of Experimental Psychology* 1935; **18**: 643-62.
- RODEN S, HARVEY P, MITCHARD M. The influence of alcohol on the persistent effects on human performance of the hypnotics mandrax and nitrazepam. *International Journal of Clinical Pharmacology and Biopharmacy* 1977; **15**: 350-5.
- NAKANO S, GILLESPIE HK, HOLLISTER LE. A model for evaluation of anti-anxiety drugs with the use of experimentally induced stress: comparison of nabilone and diazepam. *Clinical Pharmacology and Therapeutics* 1978; **23**: 54-62.
- GRIFFITHS AN, JONES DM, RICHENS A. Zopiclone produces effects on human performance similar to flurazepam, lormetazepam and triazolam. *British Journal of Clinical Pharmacology* 1986; **21**: 647-53.
- DRUMMOND, GB. The assessment of postoperative mental function. *British Journal of Anaesthesia* 1975; **47**: 130-42.
- JAMES FM. The effects of cyclopropane anaesthesia without surgical operation on mental functions of normal man. *Anesthesiology* 1969; **30**: 264-72.

13. ROBINSON JO. A study of neuroticism and casual arterial blood pressure. *British Journal of Social and Clinical Psychology* 1963; **2**: 56-64.
14. NIGHTINGALE P, PETTS NV, HEALY TEJ, KAY B, MCGUINNESS K. Induction of anaesthesia with propofol ('Diprivan') or thiopentone and interactions with suxamethonium, atracurium and vecuronium. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 31-4.
15. MIRAKHUR, RK, SHEPHERD WFI. Intraocular pressure changes with propofol ('Diprivan'); comparison with thiopentone. *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 41-4.
16. UTTING JE, FAHY L, VAN MOURIK GA. A comparison of thiopentone and propofol ('Diprivan') for induction of anaesthesia. (Abstract). *Postgraduate Medical Journal* 1985; **61** (Suppl. 3): 84.
17. HERBERT M. Psychomotor effects of anaesthetic agents. In: Smith RJ, Simpson KH, eds. *Psychology, pain and anaesthesia*. London: Harwood (in press).
18. MILLAR K. Some chronic problems in the methodology of performance tasks applied in clinical settings. In: Broughton R, Ogilvie RD, eds. *Sleep, arousal and performance*. Cambridge, (Mass); Birkhäuser, (in press).

## Peri-operative drug prescribing pattern and manufacturers' guidelines

### An audit

M. T. KLUGER, S. GALE, J. L. PLUMMER AND H. OWEN

#### Summary

*Many patients admitted for surgery are receiving regular drug therapy. Adverse effects may occur, either as a result of these drugs being stopped suddenly or because staff are unaware of significant interactions between certain drugs and anaesthetic agents or techniques. This study aimed to find out how regular drug medication is actually given in the peri-operative period. In addition, pharmaceutical companies were contacted and asked for information about the effects of sudden withdrawal of their products and potential interactions with anaesthetic agents. We found that many drugs were omitted peri-operatively with potentially significant effects. Pharmaceutical companies do not seem to appreciate the importance of this problem and not all of them give clear recommendations relevant to practising anaesthetists.*

#### Key words

*Complications; peri-operative medication.*

*Interactions, drug; anaesthetics.*

*Surgery; peri-operative period.*

In a recent survey from our hospital<sup>1</sup> it was found that the incidence of concomitant medication in patients presenting for both elective and emergency surgery rose steadily as the patients' age increased. Over 70% of patients older than 70 years of age were receiving some form of drug therapy. The nature of the drug is an important consideration in determining whether or not to stop medication; acute withdrawal of regular cardioactive medication is likely to have more serious consequences than the cessation of occasional nonsteroidal analgesic therapy. Guidelines produced by manufacturers and pharmacies for the management of patients receiving medication who present for surgery are incomplete. Information sheets supplied by pharmaceutical companies provide limited data about the effects of sudden withdrawal of a drug or its use in conjunction with anaesthesia. Two audits have been carried out, partly as a result of our first study<sup>1</sup> and partly due to the interest expressed by the Department of Pharmacy in the lack of appropriate guidance from pharmaceutical companies.

In the first study, our aim was to find out what drugs patients were taking when they were admitted to hospital, what drugs were actually prescribed by the medical staff, and if these drugs were not given, the reasons for their omission. The second study involved a survey of all pharmaceutical companies that supplied our hospital. The

purpose of this was to find out exactly what they recommended as peri-operative management for patients taking their products, and whether or not they knew of any possible interactions between the product and anaesthetic agents.

#### Methods

An audit form was designed to record the peri-operative medication prescriptions of patients, after approval from the Departments of Nursing and Anaesthesia and Intensive Care. Nursing and medical staff on the ward were not informed of the study, to prevent any alteration in normal clinical practice. The study was carried out prospectively, for two consecutive weeks. Information was obtained on the patients' current drug treatment as recorded in the medical notes, all drugs prescribed on each day of the patients' stay in hospital and reasons for not administering a dose of oral medication. Data were obtained from all adults who underwent inpatient or day case surgery.

A survey of manufacturers' recommendations was carried out by sending a standard letter to all the pharmaceutical companies which supplied the Department of Pharmacy. The following questions were asked; (a) What are the effects of sudden withdrawal of the medication? (b)

M.T. Kluger, MB, ChB, DA, FCAnaes, Senior Registrar, S. Gale, B Pharm, Senior Pharmacist, J.L. Plummer, PhD, Senior Hospital Scientist, H. Owen, MD, FFARACS, FCAnaes, Senior Staff Specialist in Anaesthesia, Department of Anaesthesia and Intensive Care and Department of Pharmacy, Flinders Medical Centre, Bedford Park, Adelaide 5042, South Australia, Australia.

Accepted 29 September 1990.

Is the drug available in a formulation other than oral? (c) What are the recommendations about substituting another drug if the one prescribed is not available? and (d) What are the possible interactions with regional or general anaesthesia?

Interest was evaluated by the response rate of the companies, the time taken to respond, information given and recommendations made that could be of benefit to anaesthetic clinical practice.

Data were analysed using SPSS-X (Statistical Package for the Social Sciences) on an Encore mainframe computer.

### Results

The records of 241 patients were reviewed. The data on 16 (6%) were incomplete so these patients were excluded from the analysis. Medication was taken by 113 patients (44%) before admission. The total number of prescriptions was 238, with an average of 2.1 drugs per patient. The main groups of drugs are shown in Figure 1. Cardioactive medication accounted for the largest proportion of prescriptions (41%). The distributions of the individual agents are shown in Table 1 for each class of drugs. Table 2 shows the number of drug doses omitted as a percentage of the number prescribed pre-operatively. Almost half (49%) of all drugs were omitted on the day of surgery, whilst on the first day after the operation one third of medications were not given. Reasons for omission of these prescriptions are shown in Figure 2. The most common reason (49%) for patients not getting their medication was that they were fasting. Thus, in the absence of prescribing guidelines, regular drug therapy was withheld by nursing staff. The second main reason for omission of concurrent medication was a failure of the admitting doctor to prescribe it.

The 45 pharmaceutical companies that supplied the hospital dispensary were contacted; after 9 months 27 (55%) had replied.

The majority of companies who replied did so within 2 to 4 weeks (58%). The time taken to respond ranged from less than 14 days (5%) to more than 7 months (4%).

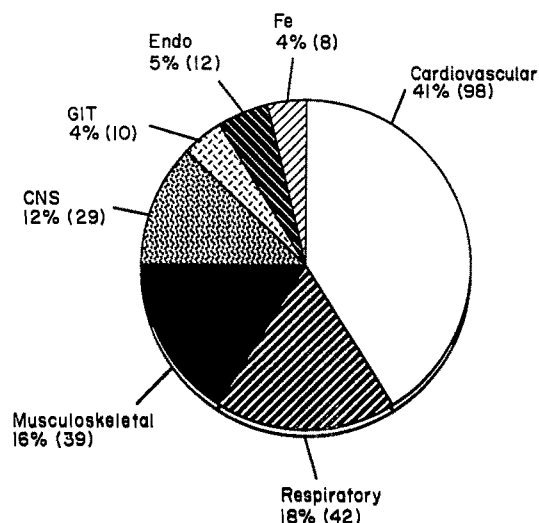


Fig. 1. Number of prescriptions broken down into major drug groups. (CNS, drugs acting on the central nervous system; GIT, drugs acting on the gastrointestinal tract; Endo, endocrine medication; Fe, iron supplements.)

Table 1. Concurrent medication: prescribed drugs.

		% of total	Number
Cardiovascular system	Betablockers	15	17
	Ca <sup>++</sup> blockers	10	11
	ACE inhibitors	7	8
	Diuretics	31	35
	Digoxin	9	10
	Vasodilators	5	6
	Other cardiovascular system drugs	4	5
Respiratory system	Salbutamol	18	20
	Ipratropium	4	5
	Beclomethasone	4	5
	Steroid	1	1
	Theophylline	4	5
	Other respiratory drugs	5	6
Endocrine	Insulin	1	1
	Oral hypoglycaemic agents	3	3
	Oral contraceptive	2	2
	Thyroxine	3	3
	Other endocrine	3	3
	Antacids	4	4
Gastrointestinal	H <sub>2</sub> blockers	5	4
	Spasmolytics	1	1
	Phenytoin	4	4
Central nervous system	Valproate	1	1
	Barbiturates	1	1
	Tricyclic antidepressants	9	10
	Analgesics	3	3
Miscellaneous	Benzodiazepines	12	10
	Non Steroidal anti-inflammatory drugs	22	25
	Allopurinol	8	9
	Antibiotics	8	9
	Fe <sup>++</sup>	4	5

In the case of nearly two-thirds (65%) of drugs the pharmaceutical companies reported potential adverse effects if the drug were to be stopped suddenly (Table 3). Forty-nine percent of these medications could be given by a

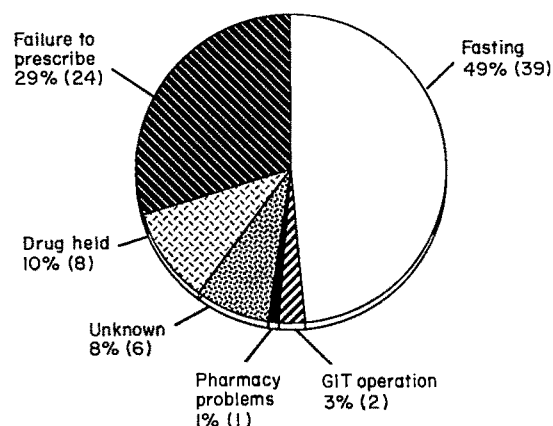


Fig. 2. Reasons for failure to prescribe concurrent medication in the peri-operative period. (GIT operation; prolonged ileus resulting in failure to institute normal feeding. Drug held; medication withheld on order by medical staff. Pharmacy problems; drug not in stock or not yet delivered to ward. Fasting; pre-operative with holding of all oral medication. Failure to prescribe; admitting medical officer did not prescribe normal medication).



**Table 2.** Omission rate on day of operation and the first day after operation. Numbers as a percentage of normal medication.\*

Drugs	% of total, operative day	% of total the first day after operation
Betablockers††	31	31
Ca <sup>+2</sup> blockers‡	34	14
ACE inhibitors‡	44	28
Diuretics††	55	28
Digoxin†	50	10
Other cardiovascular system medication	75	50
Salbutamol††	18	18
Ipratropium	33	9
Beclomethasone	13	17
Theophylline††	30	17
Other respiratory medications	71	63
H <sub>2</sub> blockers	60	25
Tricyclic antidepressants††	44	56
Benzodiazepines†	45	57
Non steroidal anti-inflammatory drugs	65	38
Antibiotics	49	33

\* Only medications prescribed for four or more patients are shown.

† Adverse effects if drugs suddenly stopped.

‡ Known interaction with local or general anaesthetic.

route other than oral. If the drug was either not available at all or could not be given by another route, most companies suggested that no replacement was necessary. Forty-five percent of drugs could potentially interact with local and general anaesthetics, whilst 30% of drugs had no known interactions with these agents.

Forty-five companies were contacted. Two showed great interest, supplying references, papers and recommendations for therapy. Both stated that they would investigate the problem further. Nine gave guidelines and a few references; 10 replied with a tabulated letter showing moderate interest; four expressed no interest but made a few superficial comments. Twenty companies did not reply at all.

### Discussion

The inception of the Audit Committee in the College of Anaesthetists has highlighted the growing awareness that important advances in our specialty can be made from critical appraisal of routine clinical practice.<sup>2</sup>

We have found that approximately two-thirds of drugs prescribed to patients who presented for surgery had potential adverse effects if they were stopped suddenly. However, over half of all medications could be given by an alternative route, so there is no reason why a greater proportion cannot be given this way in the peri-operative period.

Significant numbers of drugs taken regularly by patients have potential interactions with regional or general anaesthetic techniques. This is often not appreciated by anaesthetists nor is it emphasised by drug companies in their drug information sheets.

Wyld<sup>3</sup> reported that 15% of patients had their drugs omitted pre-operatively; Corallo<sup>4</sup> showed 67% of patients had these withheld on the day of operation, whilst a study from our own institution showed that 46% of patients undergoing elective surgery and 41% undergoing emergency surgery had their normal medication stopped pre-operatively.<sup>1</sup>

The harmful effects of stopping beta adrenergic blocking agents are well recognised,<sup>5</sup> and include arrhythmias, hypertension and myocardial ischaemia. A recent review on major aortic surgery stated that adequate pre-operative antihypertensive therapy was the most important prophylactic measure against postoperative hypertension and strongly recommended that antihypertensive medication be maintained throughout the peri-operative period.<sup>6</sup> Adverse effects have been seen when digoxin, methyldopa, clonidine and trinitrates are stopped suddenly. The withdrawal of respiratory medication, the second most commonly prescribed group of drugs, can have detrimental effects in the peri-operative period and sudden withdrawal of anti-convulsants is associated with return of seizure activity. Abrupt cessation of major tranquillisers may result in withdrawal effects, or a return of psychiatric symptoms.<sup>7</sup>

The reasons for withholding medication originate from a lack of appreciation by both nursing and medical staff of the potential problems of withdrawal. Junior resident staff, responsible for prescribing routine drugs, may fail to write up all medication the patient is currently taking. This may be a deliberate therapeutic decision, but may also be simple oversight. Adequate fasting is often interpreted by nursing staff as withholding all oral intake, including medication. Drugs are frequently given at the discretion of the nurse on duty with little guidance from medical or pharmacy staff.

Pharmaceutical companies have an important role in producing guidelines for their products. In Australia, there are approximately 1.5 million operations per year, thus potential interactions between current medical therapy and anaesthesia are frequently encountered. Although some companies do produce relevant information, a significant number showed no interest in providing information that was clinically relevant. Drug information sheets that refer to ether, chloroform and trichloroethylene clearly do not base their recommendations on current anaesthetic techniques. Some companies made very useful recommendations in response to our questions, but at the same time stated that the information supplied was not for general release.

It can be argued that these decisions are the respons-

**Table 3.** Manufacturers' data and recommendations on peri-operative medication. Actual numbers of drugs shown in parenthesis.

Adverse effect of sudden withdrawal			Alternative route of administration		Recommendations to change to another product perioperatively			Known interactions with general or local anaesthetics		
Yes	No	Not Known	Yes	No	Yes	No	Not Known	Yes	No	Not Known
65%	30%	5%	49%	51%	22%	51%	27%	45%	25%	30%
(55)	(25)	(5)	(42)	(43)	(19)	(43)	(23)	(38)	(22)	(25)

Number in parenthesis reflects individual drugs.

ibility of the anaesthetist involved in the case and that industry guidelines are of no relevance to clinical management. However, anaesthetists need information about drug interactions; textbooks often lack up-to-date information, references from journals are not always available and are time consuming to retrieve. Often the only rapidly accessible drug information is from a drug compendium e.g. MIMS (*Monthly index of medical specialities*). Drug companies have an important role in providing accurate information to guide clinicians. It is surely the responsibility of drug manufacturers to provide up-to-date information relevant to all situations in which their product may be used. The provision of such data is, however, not a requirement for a marketing licence. The low response rate and the lack of interest shown by most pharmaceutical companies who did reply, seem to suggest that they do not appreciate that anaesthesia presents a problem.

One of the aims of the College of Anaesthetists is to encourage audit of all activities of anaesthetic departments.<sup>2</sup> The audit of premedication practice carried out in this hospital<sup>1</sup> led to this present survey. It has raised several important issues pertinent to drug therapy and the guidelines for its management during the operative period. A new protocol has been devised for use in the wards partly as a result of this study. All patients now have all their medication given, unless specified by the anaesthetist during the pre-operative assessment. Nurses no longer have to make a decision about which drugs to give; this becomes the responsibility of the medical, particularly the anaes-

thetic, staff. Pharmaceutical companies need to be made more aware of potential problems related to anaesthesia. They must also accept some of the responsibility themselves, by producing useful guidelines for clinicians to follow.

#### Acknowledgments

Dr M. Kluger is employed on a grant from Janssen-Cilag Pty. Ltd (Australia).

#### References

1. KLUGER MT, OWEN H. Premedication: an audit. *Anaesthesia* (in press).
2. First Report of the Audit Committee. *College of Anaesthetists Newsletter* 1990; 3: 4-6.
3. WYLD R, NIMMO WS. Do patients fasting before and after operation receive their prescribed drug treatment? *British Medical Journal* 1988; 296: 744.
4. CORALLO CE, DOOLEY M, LOVE JB. Failure to administer prescribed preoperative drugs. *Australian Journal of Hospital Pharmacy* 1989; 19: 198-9.
5. GEORGE CF. Hazards of the abrupt withdrawal of drugs. *Prescribers' Journal* 1985; 25: 31-9.
6. CUNNINGHAM AJ. Anaesthesia for abdominal aortic surgery—a review (part II). *Canadian Journal of Anaesthesia* 1989; 36: 568-77.
7. GALLANT DM, EDWARDS CG, BISHOP MP, GALBRAITH GC. Withdrawal symptoms after abrupt cessation of antipsychotic compounds: clinical confirmation in chronic schizophrenics. *American Journal of Psychiatry* 1964; 121: 491-3.

## Doxapram after general anaesthesia

### Its role in stopping shivering during recovery

V. SARMA AND E. N. S. FRY

#### Summary

*A group of patients who developed postoperative shivering after receiving inhalational anaesthesia were assigned, at random, to receive either doxapram or a placebo under double-blind conditions. A significantly higher proportion of patients stopped shivering after being given doxapram than after the placebo.*

#### Key words

*Complications; shivering, muscle rigidity.  
Pharmacology; doxapram.*

There is a need to find a drug that can control postoperative shivering since both methylphenidate<sup>1</sup> and orphenadrine<sup>2</sup> are no longer obtainable. Doxapram, both by infusion and intravenous bolus, has been used to reduce the incidence and severity of postoperative pulmonary complications.<sup>3,4</sup> This study was designed to confirm the observation made by one of the authors that doxapram, injected at the end of general anaesthesia, prevents or curtails episodes of shivering and muscle rigidity.<sup>5</sup>

#### Methods

The protocol of the trial was approved by the hospital ethics committee.

All patients aged from 16 to 60 years, ASA grades 1 and 2, who had a general anaesthetic that included halothane, enflurane or isoflurane, were included in the trial. The first 60 patients who developed significant shivering, were placed in one of two groups (doxapram 100 mg and placebo) by means of numbered ampoules.

The age, weight, sex, and duration of surgery were recorded. Details of the anaesthetic agent, antiemetic and analgesic drugs were included. The time that shivering first appeared, the axillary temperature at that time and the presence of nausea or vomiting were noted. Patients were observed for 3 minutes to confirm the presence of significant shivering. The contents of one of the numbered ampoules were injected over 15 seconds.

After the injection of the study drug it was recorded whether or not the shivering stopped, and, if it did, the time

at which this occurred. The presence of nausea or vomiting was recorded.

If a patient continued to shiver for more than 5 minutes after the injection of the study drug, doxapram 60–100 mg was injected in the same manner as the contents of the trial ampoule. The observations were then repeated.

#### Statistical analysis

Significance level was taken as 5% throughout. Patients' ages, weights and duration of surgery were tested for differences between the groups using Mann–Whitney tests. Patients' sex, the anaesthetic agent used, antiemetic and analgesic drugs were tested using Chi-squared tests as were the time of shivering and incidence of pre-injection nausea. The temperature while shivering was tested by Mann–Whitney tests. Incidence of stopping shivering after treatment, its associated time (immediate or later) and incidence of nausea and vomiting were assessed by Chi-squared tests. The proportion of patients who stopped shivering, after the use of additional doxapram, were treated against a binomial distribution, with parameter 0.5, using a binomial test.

#### Results

Twenty-nine patients were entered into each group. Two patients were lost to the trial because one ampoule was opened in error when a patient was not shivering, and one was broken and the contents lost. Table 1 summarises the

V. Sarma, FFARCS, Consultant, E.N.S. Fry, FFARCS, Consultant, Department of Anaesthetics, North Tees General Hospital, Hardwick, Stockton on Tees, Cleveland TS19 8PE.

Accepted 5 August 1990.

**Table 1.** Patient and anaesthetic data. There was no significant difference between the two groups.

	Doxapram <i>n</i> = 29	Placebo <i>n</i> = 29
Males	14	13
Females	15	16
Age; years, mean (SD)	43.8 (15.1)	43.9 (17.8)
Weight; kg, mean (SD)	70.9 (9.4)	67.1 (12.9)
Operation time; minutes, mean (SD)	67 (38)	83 (87)
Number given halothane	4	3
enflurane	22	21
isoflurane	3	1
Mean axillary temperature (range)	36.0 (34.2–37.5)	36.0 (34.0–38.0)

**Table 2.** Effect of trial drugs on shivering, the incidence of emetic sequelae and the perioperative use of antiemetics and analgesics.

	Doxapram <i>n</i> = 29	Placebo <i>n</i> = 29	Significance
Shivering stopped	22	7	<i>p</i> = 0.0002
Time taken to stop shivering:			
< 1 minute	15	2	
> 1 minute	7	5	<i>p</i> = 0.045
Nausea	2	4	NS
Vomiting	1	1	NS
Number given antiemetics	21	23	NS
Number given analgesics	24	23	NS

NS, not significant.

ages, weights, sex, duration of operation, the inhalational agent used and the axillary temperature at the time of shivering. Table 2 summarises the response to injection of the trial drugs, the incidence of nausea and vomiting and the perioperative use of antiemetic and analgesic drugs.

### Discussion

Muscle spasticity and shivering may occur during recovery from inhalational anaesthesia especially when halothane or enflurane has been given. These are self-limiting sequelae but can be unpleasant for the patient, may last for many minutes, and can cause significant hypoxaemia.<sup>6,7</sup>

A previous study indicated that an intravenous bolus of doxapram given at the end of an inhalational anaesthetic both prevented shivering and curtailed it if it occurred.<sup>5</sup> This trial confirms the effectiveness of doxapram in stopping established shivering.

### Acknowledgments

The authors thank Mr C.J. Jones of A.H. Robins for supplying the ampoules and helping to design the trial, and

Ms C. Bye for performing the statistical analyses. We are also grateful to the anaesthetists of the North Tees General Hospital for their cooperation and especially to Sister L. Summerbell for supervising the observations in the recovery ward.

### References

1. BRICHARD G, JOHNSTONE M. The effect of methylphenidate (Ritalin) on post-halothane muscular spasticity. *British Journal of Anaesthesia* 1970; **42**: 718–22.
2. FRY ENS. Postoperative shivering. *Anaesthesia* 1983; **38**: 172.
3. GAWLEY TH, DUNDEE JW, GUPTA PK, JONES CJ. Role of doxapram in reducing pulmonary complications after major surgery. *British Medical Journal* 1976; **1**: 122–4.
4. DOWNING JW, JEAL DE, ALLEN PJ, BULEY R. I.v. doxapram hydrochloride and pulmonary complications after lower abdominal surgery. *British Journal of Anaesthesia*, 1977; **49**: 473–7.
5. FRY ENS. Muscle spasticity. *Anaesthesia* 1990; **45**: 67–8.
6. DAWKINS M. Halothane. *Lancet* 1961; **2**: 1259.
7. JONES HD, MCLAREN CAB. Postoperative shivering and hypoxaemia after halothane, nitrous oxide and oxygen anaesthesia. *British Journal of Anaesthesia*, 1965; **37**: 35–41.

## Cardiac anaesthesia in a patient with myotonic dystrophy

M. TANAKA AND Y. TANAKA

### Summary

*We describe a patient with myotonic dystrophy who required open-heart surgery for an atrial septal defect. He also had a sick sinus syndrome and an abnormal myocardium on histological examination. Anaesthesia using fentanyl, droperidol, nitrous oxide and a low concentration of enflurane was uneventful. Atelectasis of the left lung developed on the first postoperative day after removal of the tracheal tube. This was successfully treated by fiberoptic bronchoscopy.*

### Key words

*Anaesthesia; cardiac.*

*Complications; myotonic dystrophy, sick sinus syndrome, atelectasis.*

Myotonic dystrophy (dystrophia myotonica) is a generalised disease of autosomal dominant inheritance. It is characterised by myotonia, muscle atrophy, cataracts, cardiac disease, gonadal atrophy and endocrine disorders.<sup>1,2</sup> Patients may show abnormal responses to muscle relaxants,<sup>3</sup> and respiratory insufficiency can occur after surgery. We report a patient with myotonic dystrophy who underwent anaesthesia for a patch closure of an atrial septal defect (ASD) and permanent pacemaker implantation for a conduction disturbance.

### Case history

The patient was a 41-year-old male who weighed 65 kg. Cardiomegaly had been detected when he was at junior high school, but this was not investigated. He had a 3-year history of muscle weakness and myotonia of the upper limbs. Bilateral cataracts were present. The diagnosis of myotonic dystrophy had been made 2 years previously. More recently, a heart murmur and bradycardia had been noted and an ASD with sick sinus syndrome was diagnosed. There was no history of syncopal attacks. No family history of myotonic dystrophy was elicited. He was admitted for cardiac surgery with the aid of cardiopulmonary bypass (CPB). The patient was of slightly low intelligence and suffered from hypersomnia. Frontal balding, hatchet face, atrophy of sternomastoid muscles and weakness of the distal upper limb muscles were noted. Myotonia of the tongue and grip myotonia of both hands were present. He had a waddling gait with a protuberant abdomen, which suggested atrophy of the pelvic girdle muscles. He also had bilateral cataracts. Electrocardiogram

(ECG) showed right bundle branch block and episodes of bradycardia of 35 beats/minute. Holter ECG monitoring showed 69019 QRS complexes per 24 hours, and sick sinus syndrome (SSS) was suspected. There was no evidence of atrioventricular (A-V) block and no ventricular premature beats were seen. Electrophysiological studies showed a sinus node recovery time (SNRT) of 1860 msec and a corrected SNRT of 570 msec. The diagnosis of SSS was made. On auscultation, there was a systolic murmur and splitting of the second heart sound. Chest X ray showed a cardiothoracic ratio of 54%. Echocardiography and cardiac catheterisation showed mild enlargement of the right atrium and ventricle, mild tricuspid regurgitation, and an ASD. The left to right shunt ratio was 34.2%. The cardiac index was 4.2 litres/minute/square metre and the ejection fraction on echocardiography was 0.89. There were no signs of cardiac failure or mitral valve prolapse. Electromyography showed myotonic discharges and continuation of insertion voltages in all limb muscles, and a weak contraction test showed a myogenic pattern of short duration and low amplitude. No endocrine disorders were detected and testicular atrophy was not present. Pulmonary function tests showed a mixed ventilatory defect with a vital capacity of 1.93 litres (51.7% of predicted) and a forced expiratory volume in 1 second of 1.27 litres (69.7% of predicted).

The patient was premedicated with hyoscine 0.4 mg and droperidol 5 mg. At induction, diazepam 8 mg and fentanyl 0.2 mg were administered, followed by pancuronium 8 mg. The trachea was intubated without difficulty. No bradycardia was observed. Anaesthesia was maintained with 70% nitrous oxide in oxygen, supplemented with

M. Tanaka, MD, Y. Tanaka, MD, PhD, Professor, Department of Anesthesiology, Kyoto Prefectural University of Medicine, Kawaramachi-Hirokoji, Kamigyo-ku, Kyoto 602, Japan.

Accepted 15 September 1990.

0.2–1% enflurane. ECG, arterial pressure, central venous pressure (CVP), rectal and oesophageal temperatures were monitored. During CPB, mild hypothermia (31°C) was induced and fentanyl 0.2 mg, droperidol 5 mg, and pancuronium 4 mg were administered. Anaesthesia after CPB was maintained with air–oxygen supplemented with 0.2–0.8% enflurane. The ASD was 10 mm in diameter, and this defect was closed with a pericardial patch.

Dopamine 3 µg/kg/minute and dobutamine 3 µg/kg/minute were needed during withdrawal of CPB because the systolic blood pressure did not increase sufficiently with the increase in left atrial pressure. Normothermia was restored smoothly and no myotonia occurred. After CPB, circulatory stability was maintained with dopamine and dobutamine. The operation was completed by implantation of pacemaker electrodes into the pericardium. The operation time was 4 hours 15 minutes, and the CPB time was 1 hour 30 minutes. The blood loss was 480 ml. Arterial blood gas analysis after CPB showed a  $P_{aO_2}$  of 18.6–19.9 kPa on an  $F_{IO_2}$  of 0.6.

At the end of the operation no spontaneous respiration occurred and the patient did not regain consciousness. The pulse rate of 80/minute was maintained by the pacemaker which functioned in DDD mode. A pulmonary artery catheter was inserted, and he was transferred to the intensive care unit for artificial ventilation of the lungs using a Servo 900C ventilator.

Pulmonary capillary wedge pressure was 13 mmHg. Cardiac index measured by the thermodilution method was 2.5 litres/minute/square metre immediately after surgery. The patient regained consciousness after 2 hours. The tracheal tube was removed next morning when spontaneous respiration was established. The  $P_{aO_2}$  was 15.2 kPa and the  $P_{aCO_2}$  5.6 kPa on 40% oxygen. Subsequently, the bronchial secretions increased, and the arterial blood gas measurements deteriorated. Chest X ray (Fig. 1) showed atelectasis of the left lung and fiberoptic bronchoscopy was performed. Copious secretions were aspirated from the left main bronchus, and bronchial lavage was performed. Fiberoptic bronchoscopy was required on three occasions. By the second postoperative day he was able to expectorate secretions adequately without assistance. His subsequent recovery was uneventful, and the atelectasis gradually resolved.

### Discussion

Reports of anaesthesia in patients with myotonic dystrophy are not uncommon. However, to our knowledge, there has been no previous report of a patient undergoing cardiac surgery with the aid of cardiopulmonary bypass.

Myotonic dystrophy is often associated with cardiac disorders, of which cardiac conduction defects are the most common. In 1911, Griffith recognised a patient with this disease who had a slow pulse rate without heart block.<sup>4</sup> Occasionally cardiac manifestations may precede other symptoms.<sup>5</sup> A diagnosis of right bundle branch block and sick sinus syndrome had been made in our patient before surgery. An association with congenital heart disease has not been reported.

A prolonged P–R interval is the most frequently observed cardiac conduction disorder.<sup>6</sup> Interventricular conduction abnormalities, such as left anterior hemiblock and widening of the QRS complex, are also common.

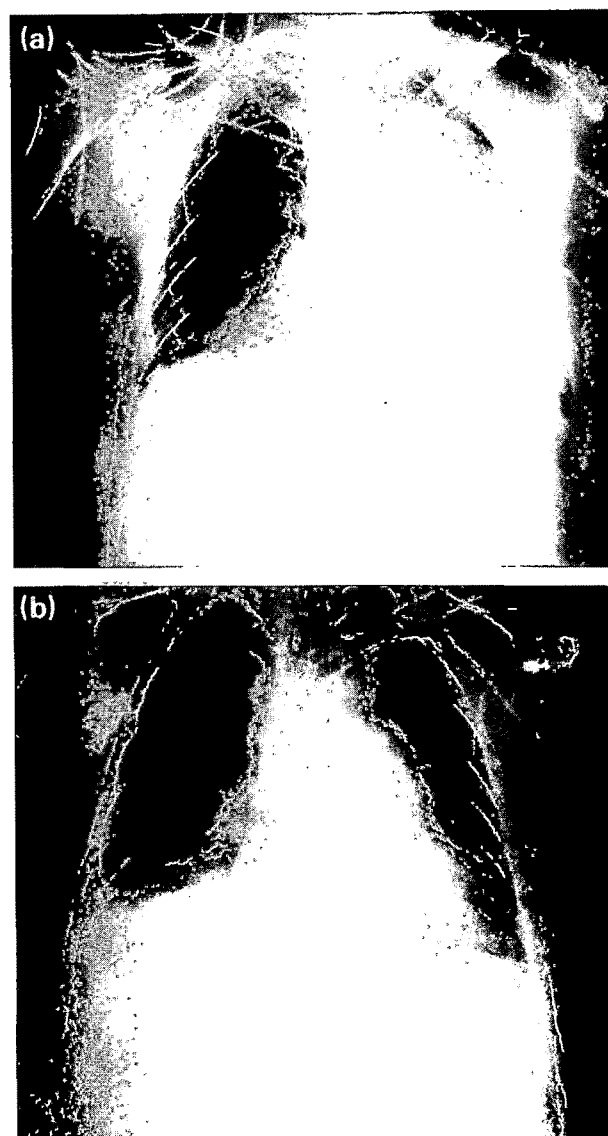


Fig. 1. Chest X rays before (a) and after (b) fiberoptic bronchoscopy.

Atrial arrhythmias such as atrial fibrillation and atrial flutter may sometimes occur. Several cases have been reported<sup>7–9</sup> in which Stokes–Adams attacks were associated with complete heart block. The more severe the disease, the greater the degree of conduction disturbance is likely to be. Therefore, when an ECG abnormality is present before surgery, electrophysiological studies may be indicated. These may show disorders of the conduction system at several sites, even when the ECG shows only P–R prolongation.<sup>10</sup> In certain cases insertion of a temporary pacemaker is recommended before surgery. In this case it was not considered necessary since no serious arrhythmias were detected pre-operatively, and in any case cardiac surgery with a right atrial approach was being undertaken.

When cardiac abnormalities are present in myotonic dystrophy, generalised degeneration of myocardium with infiltration of fatty tissue, interstitial myocardial fibrosis, irregular size of myocyte nuclei and disrupted muscle fibres are seen.<sup>8,11</sup> In this case, intra-operative myocardial biopsy from the outlet of the right ventricle showed fatty infiltration of the subendocardium, slender myocytes with irregu-

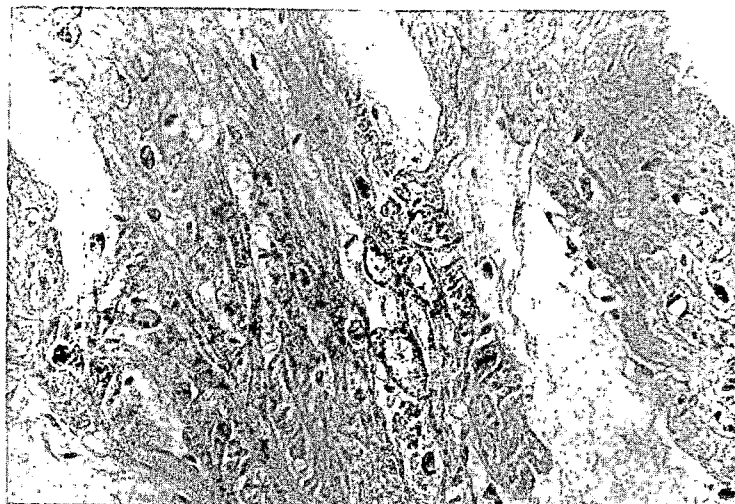


Fig. 2. Light micrograph of myocardium of right ventricle.

lar-sized nuclei, lipofuscin, and interstitial fibrosis (Fig. 2). Although in this patient there were no signs of cardiac failure before surgery, cardiac decompensation secondary to a latent cardiomyopathy has been reported during anaesthesia.<sup>12</sup> Evidence of a cardiomyopathy has been found at autopsy in more than 50% of cases of sudden death.<sup>13</sup> Anaesthetic agents with minimal effects on the cardiovascular system should be used, therefore, particularly at the time of weaning from CPB. Initially this patient required inotropic support with dopamine and dobutamine.

Respiratory complications may occur, especially during the postoperative period. Weakness and atrophy of the respiratory muscles may cause hypoventilation. Maximum inspiratory pressure and respiratory responses to hypoxia are reported to be markedly reduced in these patients.<sup>14,15</sup> In addition, somnolence and decreased cough reflexes make expectoration of sputum difficult. Respiratory failure and atelectasis have been reported in association with thiopentone, premedicant drugs and postoperative analgesics.<sup>16-20</sup> In 1959, Kaufman reported complications in nine of 25 cases.<sup>21</sup> Problems are most likely to occur when the disease is undiagnosed before operation.<sup>22</sup> Prolonged respiratory depression has also been reported.<sup>23</sup> A careful pre-operative assessment is important, as the outcome from surgery is related to the severity of the disease.

Anticholinesterase drugs can cause myotonia or may result in inadequate reversal of neuromuscular blockade.<sup>17,24</sup> If nondepolarising agents are used, it is advisable to delay removal of the tracheal tube. Shorter acting drugs such as atracurium or vecuronium have been used without reversal agents, but with careful monitoring of neuromuscular function.<sup>25,26</sup>

In this case, we electively maintained artificial ventilation until the next morning. The tracheal tube was only removed when spontaneous respiration was adequate. Despite this, collapse of the left lung occurred due to the patient's inability to expectorate the copious bronchial secretions.

Fibreoptic bronchoscopy permitted aspiration of secretions and bronchial lavage. Bronchoscopy was facilitated by the patient's inability to cough due to his primary disease.

## References

1. PRUZANSKI W. Myotonic dystrophy. A multisystem disease. Report of 67 cases and a review of the literature. *Psychiatria et Neurologia* (Basel) 1965; **149**: 302-22.
2. OKUNO T, MORI K, FUROMI K, TAKEOKA T, KONDO K. Myotonic dystrophy and hyperthyroidism. *Neurology* 1981; **31**: 91-3.
3. THIEL RE. The myotonic response to suxamethonium. *British Journal of Anaesthesia* 1967; **39**: 815-21.
4. GRIFFITH TW. On myotonia. *Quarterly Journal of Medicine* 1911; **5**: 229.
5. GRIGGS RC, DAVIS RJ, ANDERSON DC, DOVE JT. Cardiac conduction in myotonic dystrophy. *American Journal of Medicine* 1975; **59**: 37-42.
6. CHURCH SC. The heart in myotonia atrophica. *Archives of Internal Medicine* 1967; **119**: 176-81.
7. LITCHFIELD JA. A-V dissociation in dystrophia myotonica. *British Heart Journal* 1953; **15**: 357-9.
8. UEMURA N, TANAKA H, NIIMURA T, HASHIGUCHI N, YOSHIMURA M, TERASHI S, KANEHISA T. Electrophysiological and histological abnormalities of the heart in myotonic dystrophy. *American Heart Journal* 1973; **86**: 616-24.
9. CLEMENTS SD, COLMERS RA, HURST JW. Myotonia Dystrophica: Ventricular arrhythmias, intraventricular conduction abnormalities, atrioventricular block and Stokes-Adams attacks successfully treated with permanent transvenous pacemaker. *American Journal of Cardiology* 1976; **37**: 933-5.
10. KOMAJDA M, FRANK R, VEDEL J, FONTAINE G, PETITOT JC, GROSOGGEAT Y. Intracardiac conduction defects in dystrophia myotonica: electrophysiological study of 12 cases. *British Heart Journal* 1980; **43**: 315-20.
11. MOTTA J, GUILLEMINAULT C, BILLINGHAM M, BARRY W, MASON J. Cardiac abnormalities in myotonic dystrophy: electrophysiologic and histologic studies. *American Journal of Medicine* 1979; **67**: 467-73.
12. MEYERS MB, BARASH PG. Cardiac decompensation during enflurane anesthesia in a patient with myotonia atrophica. *Anesthesia and Analgesia* 1976; **55**: 433-6.
13. FEARRINGTON EL, GIBSON TC, CHURCHILL RE. Vectorcardiographic and electrocardiographic findings in myotonia atrophica: a study employing the Frank lead system. *American Heart Journal* 1964; **67**: 599-609.
14. GILLAM PMS, HEAF PJD, KAUFMAN L, LUCAS BGB. Respiration in dystrophia myotonica. *Thorax* 1964; **19**: 112-20.
15. CARROLL JE, ZWILLICH CW, WEIL JV. Ventilatory response in myotonic dystrophy. *Neurology* 1977; **27**: 1125-8.
16. RAVIN M, NEWMARK Z, SAVIELLO G. Myotonia dystrophica—an anesthetic hazard: two case reports. *Anesthesia and Analgesia* 1975; **54**: 216-8.



17. ALDRIDGE LM. Anaesthetic problems in myotonic dystrophy: a case report and review of the Aberdeen experience comprising 48 general anaesthetics in a further 16 patients. *British Journal of Anaesthesia* 1985; **57**: 1119-30.
18. BLUMGART CH, HUGHES DG, REDFERN N. Obstetric anaesthesia in dystrophia myotonica. *Anaesthesia* 1990; **45**: 26-9.
19. DUNDEE JW. Thiopentone in dystrophia myotonia. *Anesthesia and Analgesia* 1952; **31**: 257-60.
20. BRAHAMS D. Postoperative monitoring in patient with muscular dystrophy. *Lancet* 1989; **ii**: 1053-4.
21. KAUFMAN L. Anaesthesia in dystrophia myotonica: a review of the hazards of anaesthesia. *Proceedings of the Royal Society of Medicine* 1960; **53**: 183-7.
22. HARPER PS. Postoperative complications in myotonic dystrophy. *Lancet* 1989; **ii**: 1269.
23. TSUEDA K, SHIBUTANI K, LEFKOWITZ M. Postoperative ventilatory failure in an obese, myopathic woman with periodic somnolence: a case report. *Anesthesia and Analgesia* 1975; **54**: 523-6.
24. BUZZELLO W, KRIEG N, SCHLICKWEI A. Hazards of neostigmine in patients with neuromuscular disorders: report of two cases. *British Journal of Anaesthesia* 1982; **54**: 529-34.
25. NIGHTINGALE P, HEALY TEJ, MCGUINNESS K. Dystrophia myotonica and atracurium. *British Journal of Anaesthesia* 1985; **57**: 1131-5.
26. CASTANO J, PARES N. Anaesthesia for major abdominal surgery in a patient with myotonia dystrophica. *British Journal of Anaesthesia* 1987; **59**: 1629-31.

## Vocal cord paralysis in the Shy–Drager syndrome

### A cause of postoperative respiratory obstruction

P. M. E. DRURY AND E. G. N. WILLIAMS

#### Summary

*A case is presented in which unexpected and persistent postoperative respiratory problems led to the finding of bilateral abductor vocal cord paralysis and confirmed the diagnosis of the Shy–Drager syndrome. Anaesthetists should be aware that vocal cord paralysis may be a feature of this uncommon condition, and should consider the possibility of glottic obstruction as a cause of ventilatory difficulties.*

#### Key words

*Larynx; abductor paralysis.*

*Complications; Shy–Drager syndrome.*

The Shy–Drager syndrome is a rare chronic progressive disease characterised by autonomic failure and multiple system atrophy.<sup>1,2</sup> Previous reports in the anaesthetic literature have concentrated on the cardiovascular disturbances associated with this condition.<sup>3–7</sup> Vocal cord paralysis may be a feature of the syndrome, and a case is reported in which this caused complications in postoperative management and served to confirm the diagnosis.

#### Case history

A 59-year-old male patient with a 3-year history of suprapubic pain and frequency of micturition underwent an ileal loop conduit procedure for urinary diversion. Urodynamic studies had suggested a detrusor muscle malfunction without prostatic obstruction, and a trial of anticholinesterase therapy had been unsuccessful. Surgery was proposed when frequency eventually caused intolerable sleep disturbance.

He also complained of attacks of dizziness, during one of which he had fallen from a ladder and sustained a compression fracture of C<sub>6</sub>. He was a heavy smoker and had chronic bronchitis with copious sputum production. He had formerly been a heavy drinker. There was also a history of impotence.

On examination, he looked old for his years, and was undernourished. Systolic blood pressure on standing was 70 mmHg. There was weakness of the upper limbs with wasting of the small muscles of the hand. The left calf muscles were wasted. The plantar responses were extensor. The diagnosis of Shy–Drager syndrome was considered at this stage, but not pursued; a diagnosis of cervical myelo-

pathy was favoured by a consultant neurologist. Post-traumatic epilepsy was also considered.

No premedication was given. The anaesthetic technique comprised thiopentone, atracurium, tracheal intubation, nitrous oxide, oxygen, and enflurane with intermittent positive pressure ventilation (IPPV). The anaesthetic record shows occasional decreases of systolic blood pressure to 80 mmHg; these were managed successfully by fluid loading. The operation proceeded uneventfully.

The patient developed ventilatory failure 24 hours after operation. He was admitted to the Intensive Therapy Unit where his trachea was intubated and his lungs ventilated. Difficulty in weaning from IPPV was assumed to be due to retention of viscid secretions, and led to the decision to perform tracheostomy.

The tracheostomy tube was removed after spontaneous respiration had been re-established. The patient developed respiratory obstruction within 24 hours and the tracheostomy tube was re-inserted. A fiberoptic nasendoscopy was performed after two further failed attempts to remove the tracheostomy tube. This revealed bilateral abductor vocal cord paralysis with a very small airway between the cords. Further examination by a consultant neurologist revealed poor limb movement with some rigidity, and a diagnosis of Shy–Drager syndrome was made.

#### Discussion

In retrospect, the diagnosis of Shy–Drager syndrome could have been made earlier. The combination of postural hypotension and bladder dysfunction with other central nervous system signs is highly suggestive.<sup>2</sup> The possibility was raised

P.M.E. Drury, MA, MB, BChir, FFARCS, Consultant, E.G.N. Williams, MB, ChB, FFARCS, Part-time Senior Registrar, Intensive Therapy Unit, Royal Liverpool Hospital, Prescot Street, P.O. Box 147, Liverpool L69 3BX.

Accepted 5 November 1990.

at the patient's first consultation, but subsequently ignored until the finding of vocal cord paralysis. This provided an explanation for the postoperative ventilatory failure and inability to decannulate the trachea, as well as confirming the diagnosis.

The finding was unexpected, since no pre-operative history suggestive of respiratory obstruction was obtained. However, it has been reported that patients with bilateral abductor vocal cord paralysis may have normal phonation and barely audible stridor.<sup>8</sup> It is also possible that stridor is unrecognised, or misinterpreted as innocuous snoring, even by experienced observers.<sup>9</sup> This may have happened in the present case, since it seems unlikely that the vocal cord paralysis developed over 24 hours.

Vocal cord paralysis was not mentioned in the original paper by Shy and Drager,<sup>1</sup> but it has become clear subsequently that it is a feature of the syndrome, usually as a later development in the established condition.<sup>10-13</sup> It may be the presenting feature in some cases,<sup>10,14-17</sup> before the signs of multiple system atrophy develop. The Shy-Drager syndrome is uncommon, but anaesthetists should be aware of the possibility of abductor paralysis and the potential for respiratory obstruction in patients with this condition. It would be prudent to arrange an examination of the vocal cords as part of the pre-operative assessment in any patient who presents for anaesthesia and surgery and who is known to suffer from the syndrome. In our patient, the diagnosis was made easily by fiberoptic nasendoscopy.<sup>18</sup>

The vocal cord lesion is almost invariably a bilateral abductor paralysis. It is not clear why the posterior crico-arytenoid muscles (the laryngeal abductors) should be affected specifically. Studies of the muscles have revealed a picture consistent with denervation atrophy.<sup>10,19</sup> The central connection for the laryngeal abductors is known to lie in the nucleus ambiguus, but no specific central lesion has been described. However, localization of the individual laryngeal muscles in the medulla is not well defined.<sup>14</sup>

It might be expected that bilateral abductor vocal cord paralysis would be an acute emergency, but the obstruction may not always be total, and the patient's existence may be uneventful for some months after the diagnosis is made.<sup>16,20,21</sup> It is possible that muscle flaccidity may prevent an acute airway emergency.<sup>8</sup> Nevertheless, only a small additional factor, e.g. difficulty in sputum clearance, increases the risk of obstruction, as in this case.

Tracheostomy is at present the only effective treatment for bilateral abductor paralysis. A permanent tracheostomy is justifiable in the Shy-Drager syndrome, because in many cases the condition runs a relatively benign course.<sup>1,10</sup> A minitracheotomy may be useful as a temporary measure.<sup>22</sup> Sudden death has been reported in patients known to have bilateral abductor paralysis,<sup>9,21,23</sup> and consequently it is probably better to perform tracheostomy earlier rather than later. However, complex ventilatory disturbances of central origin including nocturnal apnoea and altered chemoreceptor sensitivity occur also in multiple system atrophy,<sup>7,9,11,13</sup> and may be unmasked by tracheostomy. Such patients are likely to be highly sensitive to tranquilisers and opioids.<sup>7,24</sup>

Laryngeal abnormalities are said to be present in 8% of cases of obstructive sleep apnoea,<sup>25</sup> a condition with many implications for anaesthetists and the subject of recent interest.<sup>24,26-28</sup> A proportion of these take the form of vocal cord palsies which may be a manifestation of the

Shy-Drager syndrome. Whether a wider use of nasendoscopy would reveal an increased incidence is a matter for speculation.<sup>18</sup> Consideration of obstruction at the level of the glottis should not be neglected in cases of ventilatory difficulty.<sup>29</sup>

### Acknowledgment

We wish to acknowledge the parts played by Mr A. Wolfenden, Consultant Urological Surgeon, Dr S. M. Mostafa, Consultant in Charge, Intensive Therapy Unit, Mr N. Violaris, ENT Registrar and Dr A. N. Bowden, Consultant Neurologist in the management of this patient.

### References

1. SHY GM, DRAGER GA. A neurological syndrome associated with orthostatic hypotension. *Archives of Neurology* 1960; **2**: 511-27.
2. BANNISTER R. Clinical features of autonomic failure. In: Bannister R, ed. *Autonomic failure*. 2nd edn. Oxford University Press. 1988: 267-81.
3. COHEN CA. Anesthetic management of a patient with the Shy-Drager Syndrome. *Anesthesiology* 1971; **35**: 95-7.
4. BEVAN DR. Shy-Drager syndrome. A review and a description of the anaesthetic management. *Anaesthesia* 1979; **34**: 866-73.
5. HUTCHINSON RC, SUGDEN JC. Anaesthesia for Shy-Drager Syndrome. *Anaesthesia* 1984; **39**: 1229-31.
6. MALAN MD, CRAGO RR. Anaesthetic considerations in idiopathic orthostatic hypotension and the Shy-Drager Syndrome. *Canadian Anaesthetists' Society Journal* 1979; **26**: 322-7.
7. SWEENEY BP, JONES S, LANGFORD RM. Anaesthesia in dysautonomia: further complications. *Anaesthesia* 1985; **40**: 783-6.
8. HOLINGER LD, HOLINGER PC, HOLINGER PH. Etiology of bilateral abductor vocal cord paralysis. A review of 389 cases. *Annals of Otolaryngology and Laryngology* 1976; **85**: 428-36.
9. KAVEY NB, WHYTE J, BLITZER A, GIDRO-FRANK S. Sleep-related laryngeal obstruction presenting as snoring or sleep apnea. *Laryngoscope* 1989; **99**: 851-4.
10. WILLIAMS A, HANSON D, CALNE DB. Vocal cord paralysis in the Shy-Drager Syndrome. *Journal of Neurology, Neurosurgery and Psychiatry* 1979; **42**: 151-3.
11. BANNISTER R, GIBSON W, MICHAELS L, OPPENHEIMER DR. Laryngeal abductor paralysis in multiple system atrophy. *Brain* 1981; **104**: 351-68.
12. HANSON DG, LUDLOW CL, BASSICH CJ. Vocal fold paresis in Shy-Drager Syndrome. *Annals of Otolaryngology and Laryngology* 1983; **92**: 85-90.
13. MCNICHOLAS WT, RUTHERFORD R, GROSSMAN R, MOLDOFSKY H, ZAMEL N, PHILLIPSON EA. Abnormal respiratory pattern generation during sleep in patients with autonomic dysfunction. *American Review of Respiratory Disease* 1983; **128**: 429-33.
14. MARTINOVITS G, LEVENTON G, GOLDHAMMER Y, SADEH M. Vocal cord paralysis as a presenting sign in Shy-Drager Syndrome. *Journal of Laryngology and Otolaryngology* 1988; **102**: 280-1.
15. TERÄVÄINEN H, UDD B. Vocal cord paralysis in the Shy-Drager Syndrome. *Acta Neurologica Scandinavica* 1982; **66**: 505-7.
16. LEE S, CHAN C-N. Clinical diversity of the Shy-Drager Syndrome: a case in Hong Kong. *Journal of the Royal Society of Medicine* 1989; **82**: 225-6.
17. KEW J, GROSS M, CHAPMAN P. Shy-Drager Syndrome presenting as isolated paralysis of vocal cord abductors. *British Medical Journal* 1990; **300**: 1441.
18. MCGILSHAN JA, GOLDING-WOOD DG. Snoring as a presenting feature of the Shy-Drager Syndrome. *Journal of Laryngology and Otolaryngology* 1989; **103**: 610-1.
19. GUINDI GM, BANNISTER R, GIBSON WPR, PAYNE JK. Laryngeal electro-myography in multiple system atrophy with autonomic failure. *Journal of Neurology, Neurosurgery and Psychiatry* 1981; **44**: 49-53.

20. MARTIN JB, TRAVIS RH, VAN DEN NOORT S. Centrally mediated orthostatic hypotension. *Archives of Neurology* 1968; **19**: 163-73.
21. WESTERMAN DE, FERGUSON AD. Obstructive sleep-induced apnoea due to bilateral recurrent laryngeal nerve palsies. *South African Medical Journal* 1978; **54**: 825-7.
22. HASAN A, MCGUIGAN J, MORGAN MDL, MATTHEWS HR. Minitracheotomy: a simple alternative to tracheostomy in obstructive sleep apnoea. *Thorax* 1989; **44**: 224-5.
23. LOCKWOOD AH. Shy-Drager Syndrome with abnormal respirations and antidiuretic hormone release. *Archives of Neurology* 1976; **33**: 292-5.
24. HANNING CD. Obstructive sleep apnoea. *British Journal of Anaesthesia* 1989; **63**: 477-88.
25. WILMS D, POPOVICH J, CONWAY W, FUJITA S, ZORICK F. Anatomic abnormalities in obstructive sleep apnea. *Annals of Otolaryngology, Rhinology and Laryngology* 1982; **91**: 595-6.
26. RAFFERTY TD, RUSKIS A, SASAKI C, GEE JB. Perioperative considerations in the management of tracheotomy for the obstructive sleep apnoea patient. *British Journal of Anaesthesia* 1980; **52**: 619-22.
27. TIERNEY NM, POLLARD BJ, DORAN BRH. Obstructive sleep apnoea. *Anaesthesia* 1989; **44**: 235-7.
28. HOFFSTEIN V, ZAMEL N. Sleep apnoea and the upper airway. *British Journal of Anaesthesia* 1990; **65**: 139-50.
29. ISRAEL RH, MARINO JM. Upper airway obstruction in the Shy-Drager Syndrome. *Annals of Neurology* 1977; **2**: 83.

## Anaesthesia for a patient with giant axonal neuropathy

A. MITCHELL AND P. E. MOSKOVITS

### Summary

*Giant axonal neuropathy is a generalised disorder of cytoplasmic intermediate filaments which particularly involves the peripheral and central nervous systems. In this paper we describe a child with giant axonal neuropathy and discuss the anaesthetic management in the light of the pathology and physiology of the condition.*

### Key words

*Complications; giant axonal neuropathy.*

Giant axonal neuropathy (GAN) is a rare autosomal recessive disorder of childhood.<sup>1</sup> It was first recognised in humans in 1972 and has since been described in dogs.<sup>2</sup> There is a progressive generalised disorder of cytoplasmic intermediate filaments. The peripheral and the central nervous systems are particularly involved and lesions are characterised by an accumulation of neurofilaments within swollen axons.<sup>1,3,4</sup> An early, predominantly motor neuropathy, cerebellar ataxia, pyramidal tract dysfunction, intellectual impairment, visual problems<sup>5</sup> and kyphoscoliosis are the commonest features. In most patients the hair is unusually tightly curled, emphasising the fact that abnormal intermediate filament organisation is not confined to neurones alone but may affect many other cell types. The disorder is often not detected until the third year of life, after which it is progressive, usually leading to death in adolescence.<sup>6</sup> The diagnosis is made on sural nerve biopsy.<sup>7–9</sup> More than 20 patients with GAN have been recorded in the literature,<sup>10</sup> but there is no reference to anaesthesia. We report a case and discuss the possible anaesthetic implications.

### Case history

An 11-year-old boy with moderately severe GAN presented as an emergency for left orchidectomy following acute torsion of the testis. The neuropathy had been diagnosed when he was 3-years old. A previous general anaesthetic, given for manipulation of a forearm fracture 2 years before had been uneventful. However, prolonged weakness had occurred following diazepam sedation for computerised axial tomography one year before.

On examination he was thin, with profound limb wasting and severe kyphoscoliosis, for which he wore a thoraco-abdominal brace. He had tightly curled hair and his vision was impaired, but his intelligence quotient was above average. His weight (38.8 kg) and head circumference (57 cm) were above the 50th percentile for his age, but his height (133.5 cm) was only on the 25th percentile. He had a weak cough, but no signs of a chest infection. His peak expiratory flow (PEF) was 120 litres/minute, which was normal for his height, and his arterial blood gases were also normal. He had good mouth opening and normal neck mobility. The presence of urinary incontinence suggested autonomic involvement, but as he had no evidence of cardiovascular or gastrointestinal dysfunction this was considered to be unlikely. The electrocardiogram (ECG) was normal, as was the full blood count, blood glucose, urea and electrolytes.

No premedication was given. He was monitored in the anaesthetic room with an ECG, a pulse oximeter and a noninvasive blood pressure monitor. Venous access was established. The patient was placed in a left lateral position with a head-down tilt, cricothyroid pressure was applied and anaesthesia induced with thiopentone 4 mg/kg. Anaesthesia was deepened using halothane in oxygen and the trachea was intubated with a lubricated, uncuffed tracheal tube, size 6.5. A laryngeal pack was inserted gently. No neuromuscular blocking agents were given and the lungs were artificially ventilated using halothane and nitrous oxide in oxygen. It was noted that only low inflation pressures were required. The operation was uneventful and the tracheal tube was removed with the patient awake and in a head-down left lateral position. After operation he

A. Mitchell\*, MB, ChB, Registrar, P.E. Moskovits, MB, BS, FFARCS, Consultant, Department of Anaesthetics, Kent and Canterbury Hospital, Canterbury, Kent CT1 3NG.

\*Present address: Department of Anaesthetics, King's College Hospital, Denmark Hill, London SE5 9RS.

Accepted 20 September 1990.

was given chest physiotherapy 4-hourly and monitored for 48 hours on a high dependency paediatric ward. Paracetamol 1 g 6-hourly was required for analgesia. He was discharged home on the third postoperative day.

### Discussion

This case illustrates some of the potential anaesthetic problems in a patient with GAN. Respiratory impairment associated with severe kyphoscoliosis requires careful pre-operative assessment, intra-operative support and post-operative monitoring. In general, the PEF is considered to be a good test of the respiratory pump as a whole; it is dependent on a combination of voluntary effort, muscle power, elastic recoil and airway calibre. However, the PEF values given for children are related to height, therefore their uncritical application to kyphoscoliotic patients can be misleading.

In this child, intra-operative respiratory support prevented the hypoventilation which may be associated with severe kyphoscoliosis during anaesthesia. Impairment of bulbar muscle function and a weak cough reflex may predispose patients with GAN to aspiration pneumonitis. It is therefore mandatory to protect the lungs with a tracheal tube.

Other anaesthetic techniques were considered in the light of the impairment of neuromuscular transmission. Regional anaesthesia may be technically difficult in a patient with thoracolumbar kyphoscoliosis. In addition, the inadvertent production of a high regional block could precipitate respiratory failure. However, it has been suggested that respiration in these patients is mainly dependent on phrenic nerve activity, so that interference with intercostal muscle function would have minimal effects.<sup>11</sup> Motor nerve involvement in GAN results in a decrease in nerve conduction and in acetylcholine release at the neuromuscular junction. Motor weakness occurs and the patient may be sensitive to the effects of neuromuscular blockers, inhalational agents,<sup>12</sup> particularly isoflurane and enflurane,<sup>13</sup> and sedatives. In these patients there is patchy loss of nerve function, therefore a peripheral nerve stimulator may be of limited value in assessment of recovery of the respiratory muscles from neuromuscular blockade.

Patients with denervated muscle may also exhibit sensitivity to acetylcholine, suxamethonium and anticholinesterases. In denervated muscle, a spread of acetylcholine receptors over the entire muscle membrane leads to potassium efflux on depolarisation with suxamethonium,<sup>14</sup> sometimes resulting in potentially fatal arrhythmias.

In our patient, the use of diazepam had resulted in prolonged muscle weakness. This is of significance since benzodiazepines act at the spinal cord level to reduce skeletal muscle tone, not at the neuromuscular junction.<sup>15</sup>

If the autonomic nervous system is affected, the degree of involvement is likely to be variable. Early in the disease autonomic irritability may occur resulting in hypertension and arrhythmias in response to endogenous or exogenous catecholamines. In more advanced disease, cardiovascular reflexes may be impaired, resulting in an inability to respond to hypotension produced by blood loss, vasodilatation or intermittent positive pressure ventilation. Signs of autonomic dysfunction were not evident in this patient.

### References

1. IONASESCU V, SEARBY CH, RUBENSTEIN P, SANDRA A, CANCELLA P, ROBILLARD J. Giant axonal neuropathy: normal protein composition of neurofilaments. *Journal of Neurology, Neurosurgery and Psychiatry* 1983; **46**: 551-4.
2. DUNCAN ID, GRIFFITHS IR, CARMICHAEL S. Inherited canine giant axonal neuropathy. *Muscle and Nerve* 1981; **4**: 223-7.
3. ASBURY AK, GALE MK, COX SC, BARINGER JR, BERG B. Giant axonal neuropathy: a unique case with segmental neurofilamentous masses. *Acta Neuropathologica* (Berlin) 1972; **20**: 237-47.
4. PENA SD. Giant axonal neuropathy: an inborn error of organization of intermediate filaments. *Muscle and Nerve* 1982; **5**: 1166-72.
5. KIRKHAM TH, GUITTON D, COUPLAND SG. Giant axonal neuropathy: visual and oculomotor deficits. *Canadian Journal of Neurological Sciences* 1980; **7**: 177-84.
6. DONAGHY M, BRETT EM, ORMEROD IEC, KING RHM, THOMAS PK. Giant axonal neuropathy: observations on a further patient. *Journal of Neurology, Neurosurgery and Psychiatry* 1988; **51**: 991-4.
7. CARPENTER S, KARPATI G, ANDERMANN F, GOLD R. Giant axonal neuropathy: a clinically and morphologically distinct neurological disease. *Archives of Neurology* 1974; **31**: 312-6.
8. KOCH T, SCHULTZ P, WILLIAMS R, LAMPERT P. Giant axonal neuropathy: a childhood disorder of microfilaments. *Annals of Neurology* 1977; **1**: 438-51.
9. IGISU H, OHTA M, TABIRA T, HOSOKAWA A, GOTO I, KUROIWA Y. Giant axonal neuropathy: a clinical entity affecting the central as well as the peripheral nervous system. *Neurology* 1975; **25**: 717-21.
10. MAIA M, PIRES MM, GUIMARES A. Giant axonal disease: report of three cases and review of the literature. *Neuropediatrics* 1988; **19**: 10-5.
11. LEIGH J, FEARNLEY SJ, LUPPRIAN KG. Intrathecal diamorphine during laparotomy in a patient with advanced multiple sclerosis. *Anaesthesia* 1990; **45**: 640-2.
12. NEERING I, TORDA TA. The mechanism of skeletal muscle contraction. In: SCURR C, FELDMAN S, SONI N. eds. *Scientific foundations of Anaesthesia: the basis of intensive care*, 4th edn. London: Heinemann, 1990; 516-7.
13. STOELTING RK, ed. Inhaled anaesthetic: skeletal muscle effects. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: Lippincott, 1987; 56-7.
14. KENDIG JJ, BUNKER JP, EMDOW S. Succinylcholine-induced hyperkalemia: effects of succinylcholine on resting potentials and electrolyte distributions in normal and denervated muscle. *Anesthesiology* 1972; **36**: 132-7.
15. STOELTING RK, ed. Benzodiazepines diazepam. In: *Pharmacology and physiology in anesthetic practice*. Philadelphia: Lippincott, 1987; 119-25.

CASE REPORT

## Resedation after bolus administration of midazolam to an infant and its reversal by flumazenil

S. COLLINS AND J. A. CARTER

### Summary

*A 4-month-old infant was sedated with bolus doses of midazolam, and after initial apparent complete arousal, became unresponsive and hypotonic. Administration of flumazenil enabled differentiation of a residual drug effect from an intracerebral event.*

### Key words

*Hypnotics, benzodiazepines; midazolam.  
Antagonists; flumazenil.*

Midazolam, alone or in combination with opioids is frequently used for controlled sedation on intensive care, although problems of accumulation in some patients may restrict its usefulness.<sup>1–3</sup> The benzodiazepine antagonist, flumazenil, reverses such sedation when accumulation has occurred, although the short duration of action of flumazenil may result in resedation.<sup>4</sup>

We report a case in which resedation occurred after complete arousal after boluses of midazolam. This resedation was then completely reversed by one bolus of flumazenil. This is also, to our knowledge, the first report of the use of flumazenil in an infant.

### Case history

A 4-month-old male infant, weighing 5.6 kg, was admitted to the neurosurgical intensive care unit after nonaccidental trauma, with a diagnosis of left frontal subdural haematoma demonstrated by CT scan. On arrival the child was unconscious with a tense, bulging anterior fontanelle, and his trachea had been intubated and his lungs ventilated at the referring district general hospital.

The haematoma was aspirated through the anterior fontanelle using a spinal needle, after which his conscious level improved. The decision was made electively to ventilate the infant overnight, and small doses of midazolam (0.1 mg/kg) were used for sedation, to a total of 5.5 mg administered over a 10-hour period.

The next morning sedation was stopped, and 2 hours after the last bolus of midazolam the infant was active,

breathing adequately and opening his eyes spontaneously. His trachea was extubated, whereupon he cried lustily.

However, within 45 minutes he became unresponsive and hypotonic, although respiration, as measured clinically and by pulse oximetry and arterial blood gases, was not depressed and blood glucose as measured by the Reflolux was 9 mmol/litre.

It was suspected that the haematoma had recollected; however, the anterior fontanelle was not tense, so the possible diagnosis included either a further intracerebral event or a residual drug effect. An urgent CT scan was arranged, but before this was performed 10 µg of flumazenil were administered to exclude benzodiazepine depression. Within seconds this resulted in arousal and hearty crying, with no neurological deficit apparent. A slight rise in pulse and blood pressure were observed which were consistent with the arousal of a sleeping infant. The CT scan was cancelled avoiding potential morbidity from further anaesthesia and intubation.

No further doses of flumazenil were required, and apart from a further aspiration of the subdural haematoma 6 hours later when the anterior fontanelle became tense again, the infant made an uneventful recovery.

### Discussion

It is difficult to explain why resedation after bolus doses of midazolam should have occurred. It has been reported in studies of older children or adults, who had either bolus doses or continuous infusions. Explanations based on a

S. Collins, MBBS, BMedSci, FCAnaes, Registrar, J.A. Carter, MBBS, FFARCS, Consultant in Anaesthesia and Intensive Care, Frenchay Hospital, Bristol BS16 1LE.

Accepted 3 September 1990.



second plasma peak as a result of enteral or other recirculation, or displacement of bound drug from plasma proteins or storage depots have no foundation from the plethora of current information. The 1-hydroxy-methyl and 4-hydroxy metabolites of midazolam have even shorter half lives than the parent compound, and by one hour have decreased to 10% of peak values.<sup>5</sup> As these metabolites are much less potent than midazolam, they could not be responsible for late resedation. A recent study<sup>6</sup> suggests that midazolam has a shorter elimination half-life in children than adults, and that the kinetics are possibly dose dependant, resulting in an even faster elimination in children. However, at 4 months of age, the immature hepatic enzyme systems may have delayed elimination, but one advantage of bolus dose over continuous infusion was that the drug was not allowed to accumulate, as was shown by the rapid initial awakening.

It has been reported in one study in children,<sup>7</sup> that excessive stimulation (for example tracheal suctioning) caused breakthrough agitation from otherwise satisfactory midazolam-induced sedation, and it is possible that in our case the initial arousal may have been effected by the strong sensory input of a tracheal tube *in situ*, and resedation occurred when the infant ceased to be stimulated after extubation. However, the change from complete arousal on extubation to total unresponsiveness 45 minutes later was very dramatic, as indeed was the return to full consciousness after administration of flumazenil.

This report suggests that the effects of benzodiazepines in young infants may not be predictable from their pharmacokinetics alone, and caution should be exercised in their use. Indeed, midazolam is only licenced for the induction of children over 7 years of age. However, it also demonstrates

the effective use of flumazenil to reverse benzodiazepine sedation in an infant as has previously been reported in older children and adults. Flumazenil is not licenced for use in children, but we believed that the possible avoidance of further unnecessary intervention justified its use.

## References

1. BYATT CM, LEWIS LD, DAWLING S, COCHRANE GM. Accumulation of midazolam after repeated dosage in patients receiving mechanical ventilation in an intensive care unit. *British Medical Journal* 1984; **289**: 799–800.
2. BYRNE AJ, YEOMAN PM, MACE P. Accumulation of midazolam in patients receiving mechanical ventilation. *British Medical Journal* 1984; **289**: 1309.
3. GREENBLATT DJ, ABERNETHY DR, LOCNISKAR A, HARMATZ JS, LIMJOCO RA, SHADER RI. Effect of age, gender, and obesity on midazolam kinetics. *Anesthesiology* 1984; **61**: 27–35.
4. ASHTON CH. Benzodiazepine overdose: are specific antagonists useful? *British Medical Journal* 1985; **290**: 805–6.
5. CLARKE RSJ. Biotransformation of intravenous anaesthetic agents. In: Atkinson RS, Hewer CL, eds. *Recent advances in anaesthesia and analgesia*. 14. Edinburgh: Churchill-Livingstone; 1982: 45–54.
6. SALONEN M, KANTO J, IISALO E, HIMBERG J-J. Midazolam as an induction agent in children: a pharmacokinetic and clinical study. *Anesthesia and Analgesia* 1987; **66**: 625–8.
7. SILVASI DL, ROSEN DA, ROSEN KR. Continuous intravenous midazolam infusion for sedation in the pediatric intensive care unit. *Anesthesia and Analgesia* 1988; **67**: 286–8.
8. WOOD C, ORJOT D, ROBIEUX I, DEVICOR D. Flumazénil: un antagoniste utile en pédiatrie. *Archives Françaises de Pédiatrie* 1988; **45**: 149–50.
9. BISMUTH C, BAUD FJ, FOURNIER PE, MELLERIO F. Antagonist RO 151788 dans l'intoxication volontaire par benzodiazépines, valeurs diagnostique et thérapeutique. Etude sur 40 cas. *Annales de Médecine Interne* 1986; **137**: 363–4.

## Myoclonic spasms after epidural diamorphine infusion

B. JAYAWARDENA AND D. J. HILL

### Summary

*A case is presented in which myoclonus occurred after epidural diamorphine infusion. Reports of this phenomenon following other epidural drugs and possible mechanisms are discussed.*

### Key words

*Analgesics; diamorphine.*

*Complications; myoclonus.*

*Anaesthetic techniques, regional; epidural.*

Since Behar and colleagues<sup>1</sup> described the use of epidural morphine in the treatment of pain, many opioids have been used. Side effects reported include urinary retention, pruritus and the potentially more dangerous respiratory depression. A Menière like syndrome also has been reported recently following epidural morphine analgesia,<sup>2</sup> and we report here a patient who developed myoclonus after an epidural diamorphine infusion.

### Case history

A 65-year-old woman was anaesthetised for construction of a Parks' pouch for increased frequency of bowel motions. She had been taking a high fibre diet for chronic constipation in the past. In 1974 she had an anterior resection for adenocarcinoma of her bowel and in 1988 had a colectomy with ileorectal anastomosis with a small rectal reservoir. Since then she developed increased frequency of bowel motions. She had also complained of unpleasant sensations of both legs with occasional twitching, but no abnormality was detected on neurological examination.

Previous surgery included hemithyroidectomy in 1971. She was being treated with dihydrocodeine, codeine phosphate and colofac (mebeverine hydrochloride) for her bowel complaint, and was also taking naproxen for joint pains and cervical spondylosis.

Premedication consisted of 1.5 ml of Pamergan P100 (pethidine 50 mg/ml and promethazine 25 mg/ml) given intramuscularly. Orotracheal intubation followed induction with thiopentone 250 mg, fentanyl 50 µg, vecuronium 7 mg and atropine 0.6 mg intravenously, and anaesthesia

was maintained with N<sub>2</sub>O/O<sub>2</sub> and enflurane 0.5–1% with IPPV. The patient also received prochlorperazine 12.5 mg intravenously as an antiemetic.

After induction she was positioned on her left side and an epidural catheter was inserted 4 cm at the L<sub>4/5</sub> space without difficulty. Loss-of-resistance-to-air technique was used to identify the epidural space. Following negative aspiration tests for cerebrospinal fluid and blood, 20 ml of 0.5% bupivacaine plain was injected.

The pulse rate and arterial blood pressure remained stable during the operation. Surgery proceeded uneventfully and was concluded in 3 hours; no further increments of drugs were required. When she recovered from the anaesthetic she was pain free. Preservative-free diamorphine was commenced in the recovery ward at a rate of 1.6 mg/hour through the epidural catheter and provided satisfactory pain relief. The rate of infusion was gradually reduced over the next 3 days to 0.4 mg/hour and the catheter was removed on the fourth postoperative day.

Approximately 24 hours after withdrawing the epidural catheter, the patient developed sudden episodic myoclonic spasms of both lower limbs. They were asymmetrical, more on the left than the right, and were a combination of extension and abduction at the hips and extension of the knees. These spasms caused the patient distress and were brought on by any sensory stimuli, such as touching her legs and on many occasions occurred spontaneously. Each spasm lasted 5–10 seconds and occurred at 1–2-minute intervals. There was no change in the level of consciousness and no sensory loss or pain in her lower limbs.

She was assessed at this time by a neurologist and oral clonazepam 0.5 mg three times a day was prescribed.

B. Jayawardena, MB, BS, FFARCS, Locum Consultant, D.J. Hill, MA, MB, BS, FFARCS, Consultant, Department of Anaesthetics, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ.

Accepted 14 September 1990.

Neurological examination showed exaggerated reflexes in the lower limbs with no sensory loss. The following day the myoclonia was much less frequent and less disturbing but she began to complain of jerky movement of the right arm. These myoclonic movements improved over the next few days. She was sent home on the 10th postoperative day.

Two to three days after she went home, she was completely free of the unpleasant myoclonus (uncontrollable kicking as the patient described) but the unpleasant sensations she complained of before the operation still persisted.

### Discussion

The pathophysiology of myoclonus consists of an altered discharge pattern of aggregates of nerve cells at cerebral, brainstem or spinal levels. Such independent activity could theoretically result either from removal of some inhibitory mechanisms, from direct irritation of neurones, or from a combination of such process. When the stratum of origin is interneuronal in the brainstem or spinal cord, the term is myoclonus.<sup>3</sup>

Myoclonic spasms were reported recently in a patient who received large doses of intrathecal morphine (300 µg) for pain relief as a result of malignant infiltration of lower lumbar vertebrae.<sup>4</sup> This preparation of morphine contained sodium metabisulphite as preservative.

Animal studies of the analgesic effects of intrathecal preservative-free morphine in rats regularly produced myoclonic seizures at high doses of 2 mg/kg and 4 mg/kg and these were not influenced by naloxone.<sup>5</sup> Other opioids (methadone, pethidine and fentanyl) did not produce myoclonus.

A possible mechanism by which intrathecal morphine causes myoclonic activity is by activating the 5HT system in the spinal cord. Shohami and Evron<sup>6</sup> also reported myoclonus in rats that were pretreated with para-chlorophenyl alanine methylester hydrochloride, which depleted brain and spinal cord serotonin. The morphine preparation used for their study and the diamorphine used in our patient were preservative free, excluding the possibility of myoclonus from preservatives.

Myoclonus has been reported 5 hours after a spinal anaesthetic consisting of amethocaine 14 mg with 10% dextrose in combination with adrenaline 0.2 mg. Delayed recovery of inhibitory neurones in the spinal cord was suggested as the cause.<sup>7</sup> The restless leg syndrome with altered sensations following spinal anaesthesia with 12 mg bupivacaine has been attributed to different rates of recovery of motor and sensory nerve fibres.<sup>8</sup>

Our patient received an epidural diamorphine infusion in reducing dosage over 4 days without any problems. She developed myoclonus on the fourth postoperative day 24 hours after stopping the epidural infusion. It is unlikely to be due to a direct drug action on the spinal cord; it is more likely to be an emergence phenomenon from the action of diamorphine on the spinal cord, perhaps by removal of inhibiting mechanisms.

She had complained of unpleasant tingling sensations of both legs with occasional twitching before surgery, and these symptoms persisted even when she was free from myoclonus. Therefore, the unmasking of an underlying neurological disease also remains a possibility.

### References

1. BEHAR M, MAGORA F, OLSHWANG D, DAVIDSON JT. Epidural morphine in treatment of pain. *Lancet* 1979; **1**: 527-8.
2. LINDER S, BORGEAT A, BIOLLAZ J. Meniere-like syndrome following epidural morphine analgesia. *Anesthesiology* 1989; **71**: 782-3.
3. SWANSON PD, LUTTRELL CN, MAGLADERY JW. Myoclonus—a report of 67 cases and review of the literature. *Medicine* (Baltimore) 1962; **41**: 339-56.
4. GLAVINA MJ, ROBERTSHAW R. Myoclonic spasms following intrathecal morphine. *Anaesthesia* 1988; **43**: 389-90.
5. SHOHAMI E, EVRON S, WEINSTOK M, SOFFER D, CARMON A. A new animal model for action myoclonus. *Advances in Neurology* 1986; **43**: 545-52.
6. SHOHAMI E, EVRON S. Intrathecal morphine induces myoclonic seizures in the rat. *Acta Pharmacologica et Toxicologica* 1985; **56**: 50-4.
7. FOX EJ, VILLANUEVA R, SCHUTTA HS. Myoclonus following spinal anesthesia. *Neurology* 1979; **29**: 379-80.
8. MOORTHY SS, DIERDORF SF. Restless legs during recovery from spinal anesthesia. *Anesthesia and Analgesia* 1990; **70**: 337.

## Minitracheotomy Seldinger—assessment of a new technique

I. J. B. JACKSON, A. K. CHOUDHRY, D. W. RYAN, H. R. MATTHEWS AND C. F. CORKE

### Summary

*A multicentre, prospective study of 26 patients was undertaken for the assessment of insertion of minitracheotomy tubes by the Seldinger technique. The technique of insertion is described. There were two misplacements, three blockages of the inserting Tuohy needle with fat, and six cases of difficulty in passing the minitracheotomy tube.*

### Key words

*Equipment; tubes, tracheostomy.*

Minitracheotomy, described by Matthews and Hopkinson<sup>1</sup> for the treatment of sputum retention, involves the insertion of a 4-mm flanged cannula through a vertical stab incision of the cricothyroid membrane. It permits repeated access for suction and allows oxygen entrainment. The technique is blind and problems of incorrect placement,<sup>2</sup> bleeding<sup>3-5</sup> and surgical emphysema<sup>3,4</sup> have been reported. To minimise these problems a Seldinger technique has been developed,<sup>6,7</sup> the preliminary trial of which is presented.

### Method

Five centres (four in the UK, one in Australia) experienced in minitracheotomy insertion undertook a prospective study on 26 patients with sputum retention. The Minitrach II-Seldinger kit (Portex) (Fig. 1) includes a 20-mm 16-G Tuohy needle to which a syringe could be attached, a 500-mm straight guide wire, a 70-mm 16-FG curved dilator, a 200-mm 11-FG curved introducer, a 4.0-mm internal diameter flanged tracheal cannula, a 10-FG suction catheter and a scalpel.

The patient is positioned supine with the head, neck and chin fully extended; the operator stands above the patient's head facing the patient's feet. The skin is cleansed and the position of the cricothyroid membrane located by palpation, and marked in ink. The site is infiltrated with 1-2 ml of 1% lignocaine and then massaged to diffuse the anaesthetic and restore the anatomical landmarks. A 10-mm skin incision is made and the 16-G modified Tuohy needle is inserted through the cricothyroid membrane. The embossed mark on the Tuohy needle plastic hub faces the operator, to ensure the needle bevel directs the guide wire caudally. A distinct 'give' is felt as the membrane is punctured, and correct placement is confirmed by the aspiration

of air. The guide wire is passed through the Tuohy needle into the trachea and the Tuohy needle is then removed while the position of the guide wire is maintained. The shorter, large, 16-FG dilator is railroaded over the guide wire, while firm pressure is applied. The dilator is then removed from the trachea while the guide wire is maintained within the trachea. The mini-trach cannula, premounted on the longer, small, 11-FG introducer, is then railroaded over the guide wire into the trachea, while firm pressure is applied. A gentle side-to-side screwing motion may be necessary. The introducer and guide wire are then removed while the flange of the minitrach cannula is held against the skin. It is then secured by neck tapes and suction performed. A chest radiograph including the neck, confirms its position.

### Results

Twenty-six patients were included in the study in five separate centres. Five were post-thoracotomy, one had a flail chest, two were ENT cases with partially obstructed airways and in the remainder, the indication was sputum retention.

There were two misplacements, both of which were recognised and rectified. In one of these, a minitrach tube was inserted successfully by the Seldinger technique but the guide wire could not be removed because of kinking. The second attempt resulted in misplacement. Finally, the mini-trach tube was inserted by the open method. The second patient had a very fat neck, which led to misplacement and sinus bradycardia. The Seldinger technique was abandoned and a formal tracheotomy performed.

On three further occasions the Tuohy needle was blocked with fat. Difficulties in passing the minitracheotomy tube

I.J.B. Jackson, FFARCS, Consultant, York District General Hospital, Wigginton Road, York YO3 7HE, A.K. Choudhry, FFARCS, Consultant, Sunderland District General Hospital, Kayll Road, Sunderland SR4 7TP, D.W. Ryan, FFARCS, Consultant Clinical Physiologist, General Intensive Therapy Unit, Freeman Hospital, Newcastle upon Tyne NE7 7DN, H.R. Matthews, FRCS, Consultant Thoracic Surgeon, East Birmingham Hospital, 45 Bordesley Green East, Birmingham B9 5ST, C.F. Corke, MRCP, FFARCS, ICU Specialist, Repatriation General Hospital, Heidelberg, Victoria 3081, Australia.

Accepted 5 October 1990.



Fig. 1. The Seldinger Minitrach II from Portex.

because of a shoulder between the cannula and introducer occurred in six cases. These were overcome by a side-to-side screwing motion or by repeating dilatation with the 16-FG dilator.

### Discussion

There are a variety of techniques available for patients in whom repeated access is required to treat sputum retention. These range from repeated passage of a suction catheter under direct vision into the trachea, cricothyroid installation of saline, repeated tracheal intubation and fiberoptic bronchoscopy. None are pleasant for the patient, all involve some risk of trauma and may necessitate sedation or anaesthesia. This latter could encourage a downward spiral of secretion retention, poor cough and abolished reflexes. The simplicity of the concept of minitracheotomy led Brantigen and Grow<sup>8</sup> to recommend cricothyroidotomy as an acceptable alternative to tracheotomy, with a lower risk. It is not without complications and although the Seldinger technique was proposed to reduce these, clearly misplacement, which occurred in two out of 26 patients, remains a problem. It is most likely to be promoted when correct patient positioning has failed and when entry into the trachea has been incorrectly identified, a process aided by the syringe to aspirate air. We emphasise that this kit is not suitable for patients with a very short and fat neck or in patients with a heavily calcified cricothyroid membrane. In these situations the 'open method' of insertion of minitrach II is indicated. There were no cases of haemorrhage or surgical emphysema in this study. This is in contrast with the previous technique where the blind incision of skin, cricothyroid membrane and the tissue planes between led to problems.<sup>3-5</sup> Technical difficulties were found in seven out of 26 cases; one related to the wire and six to a shoulder

created between the cannula and dilator. This problem seems to be due to one of two problems. If the original skin incision is not large enough, the skin contracts down, following insertion of the dilator. However, difficulty can also be experienced at the level of the cricothyroid membrane. Again, this is probably related to contraction following removal of the dilator. Although this was often overcome with a gentle side-to-side screwing motion, repeat introduction of the large dilator was sometimes necessary. Possible further developments would be to taper the end of the cannula, increase the size of the indwelling introducer, thus decreasing the shoulder height, or increase the size of the dilator. Portex are now marketing this product in addition to the current minitrach II, thus giving greater choice to the clinician. We believe it represents a step forward in increasing the safety of the minitrach technique.

### Acknowledgements

We are grateful to Portex Limited for their support and for supplying the 'Minitrach II-Seldinger' clinical trial sets, and to Mrs J. Mills for secretarial assistance.

### References

1. MATTHEWS HR, HOPKINSON RB. Treatment of sputum retention by minitracheotomy. *British Journal of Surgery* 1984; **71**: 147-50.
2. RYAN DW. Minitracheotomy. *British Medical Journal* 1990; **300**: 958-9.
3. PEDERSON J, SCHURIZEK BA, MELSEN NC, JUHL B. Minitracheotomy in the treatment of postoperative sputum retention and atelectasis. *Acta Anaesthesiologica Scandinavica* 1988; **32**: 426-8.
4. WAGSTAFF A, SPARLING R, RYAN DW. Minitracheostomy. *Anaesthesia* 1987; **42**: 216.

5. TERRY RM, COOK P. Haemorrhage during minitracheotomy: a reduction of risk by altered incision. *Journal of Laryngology and Otology* 1989; **103**: 207-8.
6. CHOUDHRY AK, JACKSON IJB. Minitracheotomy—a report of a proposed further development. *Annals of the Royal College of Surgeons of England* 1988; **70**: 239-40.
7. CORKE C, CRANSWICK P. A Seldinger technique for minitracheostomy insertion. *Anaesthesia and Intensive Care* 1988; **16**: 206-7.
8. BRANTIGEN CO, GROW JB. Cricothyroidectomy: elective use in respiratory problems requiring tracheotomy. *Journal of Thoracic and Cardiovascular Surgery* 1976; **71**: 72-81.

## Evaluation of the Lamtec anaesthetic agent monitor

S. J. E. HUMPHREY, N. P. LUFF AND D. C. WHITE

### Summary

*The Lamtec agent monitor is a compact anaesthetic analyser designed to measure halothane, isoflurane and enflurane. It shows good linearity and stability. The faster model can be used for end-tidal measurements up to 25 breaths per minute. Calibration using a standard of the gas to be measured is recommended.*

### Key words

*Equipment; analyser.*

The Lamtec Agent Monitor (Fig. 1) is an anaesthetic analyser designed to measure halothane, isoflurane and enflurane over the range 0–9%. It is a compact instrument (19 × 11 × 7 cm) which can be mounted on the anaesthetic machine with an optional screw clamp. The instrument is available in two versions with different specified response times (0.8 and 0.3 sec.). For this evaluation we were loaned four instruments with the faster response time (type S).

The principle of operation is similar to the Engstrom Emma.<sup>1</sup> The sensor contains two piezo electric crystals oscillating at their resonant frequencies. One crystal is a reference, the second is coated with a substance which adsorbs anaesthetic agents. The increased mass resulting from the adsorption of the agent causes a change in frequency in direct proportion to the agent concentration.

Unlike the Emma the Lamtec is a 'sidestream' analyser. The sample is pumped through the sensor chamber at a rate of about 250 ml/minute, entering and leaving via Luer taper connectors on the front of the instrument. Connexion to the anaesthetic system is through a special nonagent-absorbing tubing. Hydrophobic bacterial filters are fitted to the inlet and outlet ports; these protect the sensor from moisture.

On the front panel of the instrument are the power switch, zero and gain controls, an agent selector switch and thumbwheel switches to set high and low alarm levels. The agent concentration is shown with a resolution of 0.1% on a digital display which also shows the agent selected and any active alarm.

The instrument is powered by a 12-volt supply from a separate mains adaptor. On the back of the instrument are

sockets for the power connector and output connector. The following signals are available at the output connector: agent concentration (200 mV/%), agent selected, alarms and remote reset.

To achieve the best resolution for the purpose of evaluation, all readings were taken with a digital voltmeter connected to the voltage output on the back panel.

### Evaluation of performance

#### Linearity

Each of the analysers was connected in turn to sample the output gas stream from a vaporizer, with the analyser output connected to the sample input of a gas chromatograph with a flame ionisation detector (Pye 104). The vaporizer setting was adjusted to produce a range of anaesthetic mixtures between 1 and 5% and the analyser and gas chromatograph readings were recorded. Samples of anaesthetic standard were used to check the gas chromatograph at the start and end of the tests. The test was repeated with halothane, enflurane and isoflurane. A regression line was calculated for the results of each analyser. The slope for each instrument was adjusted for slightly different gain settings to show linearity on a single curve. Figure 2 shows the plot of analyser reading against gas chromatograph reading for the four analysers for isoflurane. For all four analysers the regression coefficient for the line of best fit was between 0.9999 and 1.0000.

Similar results were obtained for halothane and enflurane.

S.J.E. Humphrey, BA, Higher Professional and Technical Officer, Division of Bioengineering, N.P. Luff, BSc, MIBiol, CBiol, Higher Scientific Officer, Division of Anaesthesia, D.C. White, MB, BS, FFARCS, Consultant, Department of Anaesthesia, Northwick Park Hospital and Clinical Research Centre, Watford Road, Harrow, Middlesex HA1 3UJ.

Accepted 26 August 1990.



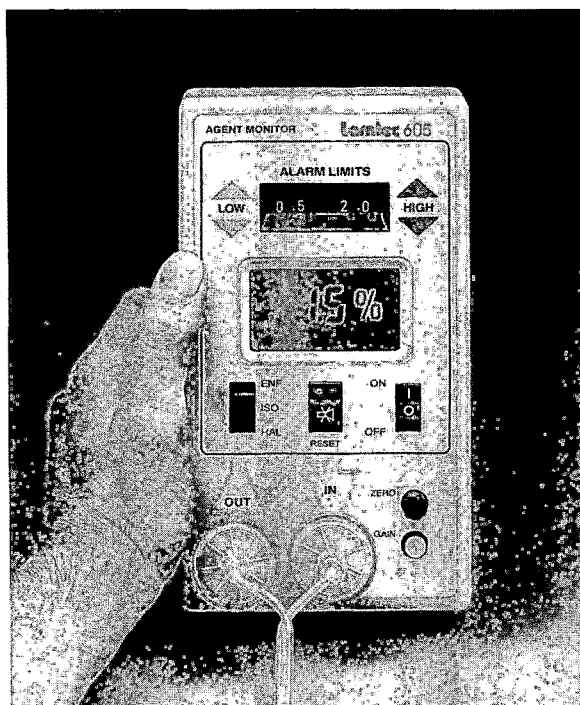


Fig. 1. The Lamtec anaesthetic analyser.

### Stability

Stability was examined for both gain (span) and zero. The tests were carried out in laboratory conditions with dry gases. We did not look at the possible effects of other gases (water vapour, nitrous oxide or carbon dioxide) on stability. An analyser was set up to sample halothane from a vaporizer with a low flow of nitrogen (2 litres/minute) as carrier gas. The vaporizer was set to 1% agent. The test continued for 4 days; during this time the analyser zero and gain were checked from time to time.

(1) *Zero*. The analyser zero was initially offset to 0.05% so that changes up and down would be seen. To check the zero the analyser was allowed to sample air for a few minutes and the voltage at the output socket was measured with a digital voltmeter.

(2) *Gain*. Readings from the analyser for samples of the same standard were taken at the start, end and after the first day. The readings, taken with a digital voltmeter

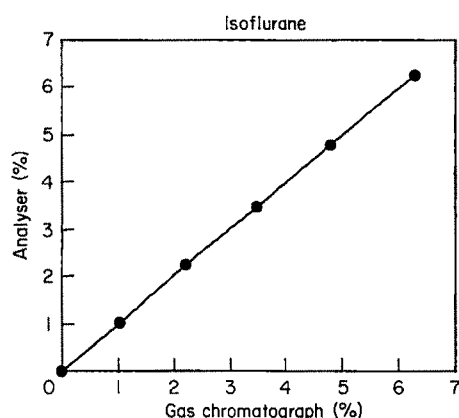


Fig. 2. Plot of analyser readings against gas chromatograph values to show linearity for four instruments.

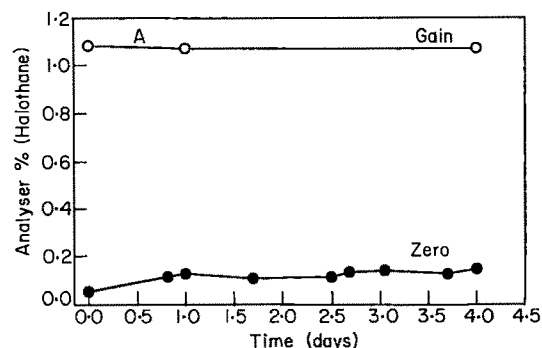


Fig. 3. Plot of gain and zero for one analyser to show stability.

connected to the output socket, were corrected for barometric pressure and analyser zero.

Figure 3 shows a plot of the gain and zero of the analyser over a period of 4 days. The change in gain was 1.0% of the reading (0.01% agent). The zero showed a drift of 0.08% agent in the first day and a subsequent variation of (SD) 0.02% agent.

The analysers all showed an initial fall in zero, measured at the output socket, after switch on. This was of the order 0.025–0.05% agent. Attempts to find a pattern in this were inconclusive. The changes sometimes occurred in 5–10 minutes and sometimes over a period of hours.

### Change between agents

The calibration kit supplied by the manufacturer contains only one agent (enflurane). In view of this it is important that setting the span for this agent should also set the span for the other agents.

To test this the output of each analyser was checked using standard mixtures of about 1% for halothane, enflurane and isoflurane. The standards were prepared in the laboratory and checked using a gas chromatograph against volumetric standards. The methods used were as described by Luff and White.<sup>2</sup>

Table 1 shows the relative error in reading for halothane and isoflurane for the four analysers after calibration using enflurane.

### Nitrous oxide

An analyser was set up to sample 100% oxygen. This was then changed to 70% nitrous oxide in oxygen and resulted in a reading of 0.08% agent.

### Response time

Response time of a sidestream analyser will be influenced by many factors. These include inherent response of sensor,

Table 1. Error in calibration (% of reading) with halothane and isoflurane after calibration with enflurane.

Analyser	Halothane (%)	Isoflurane (%)
002001	–10.8	–4.3
03255	–7.5	–6.1
03256	–8.6	–5.5
03257	–9.2	–7.9

**Table 2.** Response time (10–90%) for halothane with 1.5 m sample pipes and filters connected.

Instrument	Response time (10–90%) (msec)		Sample flow (ml/minute)
	Mean	SD	
002001	620	0	261
03255	836	9	203
03256	764	11	220
03257	844	14	198

volume of sensor chamber, sample flow rate, mixing in sample pathway, absorption of anaesthetic in the sample pathway, circuit adaptors and the method of measurement. For this evaluation we have studied the analyser in two configurations, (1) as specified for normal clinical use and (2) attached to a minimum system to indicate the inherent analyser response.

Response time was measured using a valve and recording system developed for this purpose.<sup>2</sup> The valve switches the sample line between two gas streams and the output voltage from the analyser is digitised, stored and analysed with a computer. The response time shown in the results is the time for the output waveform to change between 10 and 90% of a step change in gas concentration applied to the end of the sample input tube. Each of the results shown is the mean and SD of five measurements. The time resolution for a single measurement was 20 msec.

Table 2 shows the measured response times using halothane for a step change from 1% to 0% agent applied to the analysers with the normal input and output sample lines and the hydrophobic filters fitted. These times are much longer than the response time specified by the manufacturer for the instrument (type S). Response time is influenced by sample flow rate and by the inclusion of the input filter in the sample line. The variation in Table 2 of the response time between the four instruments is largely accounted for by the different sample flow rates.

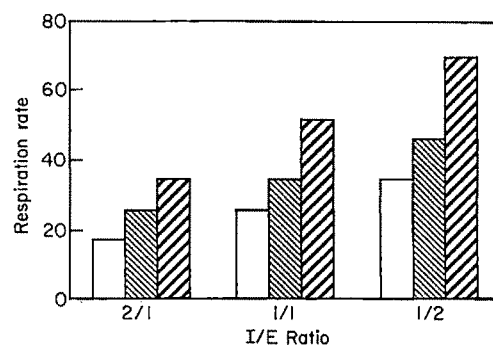
Table 3 shows the results for the same analysers with no filters, a short input sample line and a wide (4 mm internal diameter) output line. These results show reasonable agreement with the manufacturers specified response time (300 msec).

Only a small difference in response time (2% of reading) was found between the three agents tested on one analyser.

The accuracy with which an analyser can measure inspired and end-tidal concentrations in a respiratory waveform can be tested using a square wave input.<sup>1,4</sup> The method uses a valve to apply repeated step changes in gas

**Table 3.** Response time (10–90%) for halothane with 50 cm sample pipe and no filters.

Instrument	Response time (10–90%) (msec)		Sample flow (ml/minute)
	Mean	SD	
002001	284	9	378
03255	360	0	283
03256	292	11	299
03257	316	17	299

**Fig. 4.** Error in analyser readings (% of input step) for applied square wave input signals at three I/E ratios. □, 5% error; ▨, 10% error; ▩, 20% error.

concentration to the analyser input. Figure 4 shows the maximum respiration rate and error (% of signal change) in end-tidal values for analyser 03256 with pipes and filters, for 3 I/E ratios.

These respiration rates are for a square wave signal applied to the analyser. In practice the errors would be greater since the wavefront would not be square. For example, for the analyser in Fig. 4 a waveform with a front edge ramp taking 20% of the time of the step would add 1.5 and 3.5% to the error for a 1/1 I/E ratio at 25 and 50 breaths/minute.

#### Sampling system

The sample pathway consists of the circuit adaptor, the sample pipes and the analyser including the measuring chamber and sample pump.

#### Sample pipe

The pipes supplied are a flexible twin path tubing 155 cm long and 1.6 mm internal diameter selected to be nonagent absorbing. For constant anaesthetic concentrations we could not detect any difference in a standard anaesthetic mixture measured with the gas chromatograph connected directly or through the sample pipe.

There is a small effect on the response time. If the sample pipe length is doubled the response time is increased by more than can be accounted for by the change in sample flow. This was tested on one instrument fitted with an input pipe and filter but no output pipe. The results are shown in Table 4 for analyser 002001 using halothane.

The increase in response time (10–90%) is 65 msec more than can be accounted for by the change in sample flow. A similar result was obtained with a 1.5 mm internal diameter manometer line. The effect may be due to extension of the wavefront in the pipe or to absorption of agent in the tube.

**Table 4.** Effect of sample pipe length on response time.

Input pipe	Response time (10–90%) (msec)		Sample flow (ml/minute)
	Mean	SD	
1.5 m	524	9	301
3.0 m	616	9	288

**Table 5.** Effect on sample flow of water in filter.

Volume of water (ml)	Flow (ml/minute)
0	264.8
0.1	261.9
0.2	257.8
0.3	Alarm sounded Filter blocked

### Filters

The instrument handbook specifies that the input and output filters should always be used. The effect of the filters on response time has been noted above. The filters prevent water as liquid from reaching the sensor and any water in the sample line will be trapped. The filter has a small internal volume and when this is filled with water the flow is totally obstructed.

Table 5 shows the effect on sample flow of injecting water into the end of the sample line with filter in place.

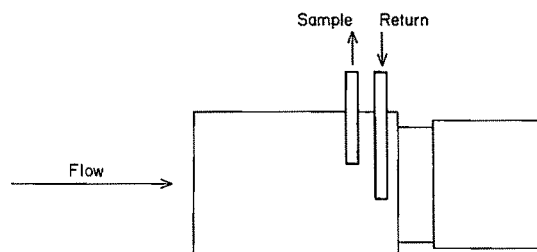
### Circuit adaptors

Three circuit adaptors, which allow the connexion of sample and return pipes, are available for the instrument. These are patient circuit adaptor, fresh-gas adaptor and Y-piece adaptor.

It is possible for the return sample to mix with the outgoing sample sufficiently to affect the response time with some configurations of the connexions. This is particularly noticeable at low gas flows. If the analyser is being used to measure end tidal values with the patient circuit adaptor or Y-piece adaptor, with the sample return to the adaptor, the return pipe should be connected after the sample pipe in the direction of flow and should be connected to the longer of the two connections (see Fig. 5).

### Sample flow rate

The effect of sample flow rate on response time was tested on one analyser with a standard input pipe and filter. The sample flow was changed by restricting the output path. The flow was measured with a bubble flow meter and the



**Fig. 5.** Suggested configuration for circuit adaptor to avoid degradation of response time at low gas flows.

response time recorded over a range of flows. The results show an inverse linear relationship between the response time and the flow.

For analyser 002001 with halothane the relationship for response time (10–90%) was:

$$T_{(10-90)} = 160900/(\text{Flow}) + 17.3$$

Response time in msec.  
Flow in (ml/minute)

correlation coefficient = 0.998

The alarm sounded for obstructed sample line when the flow had reduced to 140 ml/minute and the output pressure was 150 cmH<sub>2</sub>O above ambient pressure.

### Comments

The response time of the analyser (model S) makes it well suited to measurement of end-tidal anaesthetic concentration in adults whose lungs are artificially ventilated, but the digital display is not updated sufficiently often to follow these changes. If the analyser is to be used as a stand alone monitor for end-tidal values it would be necessary to attach a separate meter, preferably with peak and trough hold.

The analyser was generally easy to use with the exception of the zero setting control which had a large backlash and was oversensitive. We noticed during use that the voltage output had a commendably low electrical noise level compared to most infrared analysers we have seen. This is particularly noticeable with respect to halothane.

### Conclusions

The Lamtec Agent Monitor is a reliable instrument entirely suitable for clinical use. The stability and linearity of the sensor is impressive but if a high degree of accuracy is required, calibration should be carried out with the agent selected. The inherent speed of response of the sensor is impaired by the filter. Observation of end-tidal values requires the use of a meter or recorder attached to the analogue output.

### Acknowledgments

We are grateful to the manufacturer ICOR AB, Ulvsundavagen 178B, 161 30 Bromma, Sweden for the loan of four instruments for this evaluation. The 'Lamtec Agent Monitor' is available in the UK from pneuPAC Ltd, Anaesthesia Division, Norton Road, Stevenage, Herts SG1 2BB.

### References

1. LUFF NP, WHITE DC. Evaluation of the EMMA anaesthetic gas monitor. *British Journal of Anaesthesia* 1981; **53**: 1102.
2. LUFF NP, WHITE DC. Evaluation of the Datex 'Normac' anaesthetic agent monitor. *Anaesthesia* 1985; **40**: 555–9.
3. HUMPHREY SJE, WHITE DC. Measurement of response time of gas analysers. *British Journal of Anaesthesia* 1989; **63**: 227P.
4. McPEAK H, PALYWA E, MADGWICK R, SYKES MK. Evaluation of a multigas anaesthetic monitor: the Datex Capnomac. *Anaesthesia* 1988; **43**: 1035–41.

## Evaluation of the Graseby PCAS

### A clinical and laboratory study

I. J. B. JACKSON, P. SEMPLE AND J. D. STEVENS

#### Summary

*The Graseby patient-controlled analgesia system was evaluated in the laboratory and in clinical use. The problems encountered with eight examples used to treat 510 patients are reported. Laboratory performance revealed the unit to be accurate at infusion volumes of 1 and 2 ml.*

#### Key words

*Pain; postoperative.*

*Equipment; infusion pumps.*

The principles of patient-controlled analgesia (PCA) using intravenous drugs were established over 20 years ago,<sup>1</sup> when it was used to study aspects of postoperative pain and analgesia. Up until 1984 there were only two suitable infusion pumps commercially available in this country, the Cardiff Palliator<sup>2,3</sup> and the On-demand Analgesia Computer.<sup>4</sup> Recent advances in microprocessor control of medical equipment are now providing the clinician with a larger choice of pumps,<sup>5</sup> which are increasingly compact and user-friendly; they offer more control of infusion and bolus dose variables. The setting up of an acute pain service in which PCA was to play a major role led us to evaluate the accuracy and reliability of one of the latest additions to this field, the Graseby PCAS (Graseby Medical Ltd, Watford, Herts).

#### Description of Graseby PCAS

The Graseby PCAS (Fig. 1), a development of this company's syringe pump, allows the choice of infusion, intermittent bolus or a combination of these techniques, to be used. The unit, which weighs 2.75 kg, measures 16.5 × 38.0 × 9.0 cm, and can stand alone or be attached to an intravenous stand, utilises Becton Dickinson 50 ml syringes; the choice of delivery system is left to the operator. No mechanism locks the unit to the stand, but security is provided by a key-operated power/security switch that locks both the syringe in place by use of a metal cage and the dosage settings. Further control of the unit is provided by seven touchswitches and a two-line alphanumeric liquid crystal display (LCD). The upper two touchswitches

control adjustment of the various parameters (Table 1), which the two, labelled 'verify' and 'reset', call up to the LCD display to allow their adjustment. The two labelled 'run' and 'stop', control the starting and stopping of the pump and when the pump is running a green light glows on one corner of the 'run' button. This glows constantly when the unit is ready to provide the patient with a bolus. During delivery of the bolus the light flashes and is then extinguished until the lockout period has elapsed. It is possible to stop the unit temporarily by pressing the 'run' button despite the security control being in the 'on' position. The unit warns the user of this by an intermittent bleep, and when restarted continues at the point it was stopped. The final touchswitch marked 'alarm' allows resetting and muting of alarm functions. The LCD displays both status and alarm messages. The handset consists of a pneumatic plunger linked by a length of lightweight, clear, plastic tubing to the pressure sensor housed in the pump. The button is designed to be flush with the top of the handset and requires a marked depression to initiate a demand, thus decreasing the risk of accidental triggering. All patient demands are acknowledged by an audible tone.

#### Methods

The laboratory evaluation was carried out on one unit loaned by the manufacturer.

#### Laboratory assessment

*Electrical safety.* The equipment was tested to BS5724 requirements<sup>6</sup> using a Rigel 232 electrical safety tester.

I.J.B. Jackson, MB, ChB, FFARCS, Consultant, P. Semple, MB, BS, FFARCS, Registrar, J.D. Stevens, MB, BS, FFARCSI, Research Registrar, Department of Anaesthesia, York District Hospital, Wigginton Road, York YO3 7HE.

Accepted 30 October 1990.

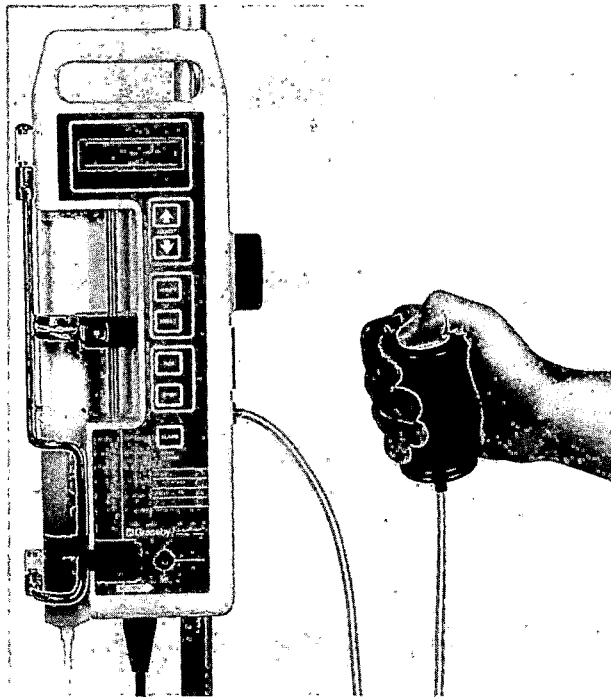


Fig. 1. The Graseby PCAS.

**Accuracy of bolus dose and infusion rate.** The accuracy of bolus dose was measured gravimetrically 10 times at four volume settings (0.1, 0.5, 1.0 and 2.0 ml), chosen as those likely to be used clinically. The infusion rates chosen were 0.5 and 1.0 ml/hour; these were tested with water five times at each setting.

**Self-contained power supply.** After being charged for 24 hours the unit was operated using its infusion mode set at 4 ml/hour until a low-battery message appeared.

**Occlusion alarm testing.** The unit was tested in the bolus dose mode with a simulated obstruction at the distal end of a 150 cm length of infusion line. The unit was operated at three different dose settings (0.1, 0.5 and 1.0 ml) until the occlusion alarm was triggered. The number of doses given and the volume infused (according to the unit) was noted on five occasions at each setting.

#### Clinical assessment

Eight pumps have been used regularly by the Acute Pain Service over a year. The service was initially introduced to six surgical and two gynaecological wards, which allowed nursing staff to be trained in their use and monitoring. During this period 660 patients were admitted to the service and of these 510 received PCA. The other 150 patients received epidural infusions which were often administered by utilising the background infusion mode of the Graseby

Table 1. Parameters that can be adjusted on Graseby PCAS.

Bolus	mg
Dose duration	stat (100 ml/minute or 5–15 minutes)
Lockout interval	3–40 minutes
Concentration	10 µg/ml to 99.5 mg/ml
Background infusion	ml/hour
Loading dose	µg or mg

Table 2. Accuracy of Graseby PCAS.

Prescribed dose (ml)	Mean	Standard deviation	Range
0.1	0.085	0.012	0.0638–0.1
0.5	0.467	0.034	0.3967–0.5051
1.0	0.994	0.032	0.9401–1.0451
2.0	1.990	0.015	1.9670–2.0158
Infusion rate (ml/hour)			
0.5	0.472	0.028	0.4221–0.4911
1.0	0.984	0.026	0.9477–1.0187

PCAS pump. Patients were established on PCA in the recovery room or on the ward by the anaesthetist using a standard morphine mixture of 1 mg/ml. The PCAS pump was set to deliver a bolus dose of 1 mg with a lockout time of 5 minutes. The dose was delivered in the 100 ml/minute (stat) mode and no background infusions were used. The unit was connected to the patients' intravenous line with a 150 cm extension line (lectro-cath, Vygon) and a Y connexion piece that contained two nonreturn valves (LS-2 Verbindungsstueck, Braun Medical). The average duration of treatment was 2 days. All the pumps were numbered and during the trial period all faults were documented.

## Results

### Laboratory assessment

**Electrical safety.** The pump is manufactured to category BF (Class II) and complies to BS5724.

**Accuracy of bolus dose and infusion rate.** The unit failed to deliver the bolus volumes prescribed as shown in Table 2. In fact the pump only performed within the manufacturers stated tolerances (1%) at the larger volumes, 1 ml and 2 ml. In the infusion mode the unit again failed to deliver the volume prescribed.

**Self-contained power supply.** The unit functioned on batteries for 12 hours on its infusion mode.

**Occlusion alarm testing.** The results of the testing of the occlusion alarm are shown in Table 3. As expected of a pressure-activated occlusion alarm it was triggered at a similar volume of infusate at all the tested bolus sizes.

### Clinical assessment

The Graseby PCAS unit developed a number of faults (Table 4) during the trial period. Many of these occurred during storage or transport and led to problems setting the pump for use; the others were potentially more serious in

Table 3. Occlusion alarm testing.

Prescribed dose (ml)	Mean number of presses	Mean volume infused (ml)
0.1	12	1.26
0.5	3	1.26
1.0	2	1.28

**Table 4.** Faults during use of Graseby PCAS.

---

Failure of erasable programmable read only memory (EPROM) package to store variables.
Handset failure.
Battery failure.
Failure of metal cage hinges.
Power lead dropping out.
Occasional loss of programme when reconnected to mains.
Supply after running on battery.
Unable to programme at start of treatment.

---

that they occurred whilst the unit was actually in patient use. The erasable programmable read only memory (EPROM) failure to retain settings has occurred on three occasions. In one case it was undoubtedly linked to the unit being dropped and in the others it was because of a fault detected in the touchswitches by the unit during the self check it performs when switched on. The units were returned to Graseby for repair after these failures. The handset problems were similarly linked to accident damage; the handsets have repeatedly been dropped on the floor during transport, storage or when setting up the pumps. This can result in the white button being released from the base plunger mechanism. Usually repair is possible by re-inserting the button and pushing it home firmly; however, three buttons have required replacing. The manufacturers assure us that the handset has now been modified and is more robust; it otherwise appears to be of successful design. Patients find it easy to use; it has a velcro strapping to help keep it in place and there are no electrical connexions, thus reducing concern about fluid spillage.

Battery failure was linked to the testing of length of operation on battery. The unit was operated in the laboratory until the alarm indicated the battery was low. It was then left without recharging for a period of some days. Upon return the unit would not function either on mains or battery use. The batteries required replacement and were found to be of a type that must not be allowed to become completely discharged; this may be important in the clinical situation.

The failure of the metal cage occurred twice in the trial period. This was the result of loosening of small studs which hold the cage to the hinges and was cured simply by tightening these with a small Allan key.

During the year as nursing staff became more familiar and enthusiastic with PCA, patients were encouraged to keep their pumps for longer and to mobilise while still using them. This led to the power supply lead being repeatedly removed and replaced from the base of the unit. Two problems have been highlighted by this practice. The first is that the plug connecting to the base of the unit can become loose and fall out during use. This problem has been previously reported.<sup>8</sup> More serious, however, is the loss of programming that can occur when replacing the power supply lead to the unit. This has happened five times and although each unit involved has shut down each time and thus has failed safely, it is a more worrying fault. Graseby have now modified the earthing of the unit and assure us that this will cure the problem. Finally, one pump failed completely when a bag of fluid situated above it on a drip stand leaked. The pump is not waterproof and care should be taken to avoid spillage of fluids onto it.

## Discussion

The pump failed to deliver the prescribed volumes but performed within the manufacturers tolerances at the larger dose settings (1 and 2 ml) which were used clinically. The performance of the unit compared favourably with previously tested units<sup>5</sup> particularly as these were tested at larger bolus volumes. Use of larger dose settings (0.5 ml and above) is not only to be recommended because of the error at lower volumes, but also in view of the function of the obstruction alarms. These are triggered by the pressure build up in the intravenous line and therefore will function earlier when larger dose volumes are utilised. One limitation of the Graseby PCAS is that control of the maximum dosage the patient can administer is obtained by setting the dose(mg) and the lockout interval controls. Theoretically this is less satisfactory than a 1- or 4-hour limit control found on some newer units. To avoid the possibility of the patient overdosing, a lockout interval of greater than 20 minutes is necessary. However, in clinical practice this has not been a problem and we have gradually reduced the lockout interval to 5 minutes and experienced few problems.

The pumps have been found to be 'user friendly' by both nursing and medical staff. Alarms are comprehensive and easily understandable with messages appearing on the LCD display. The self-contained power supply provides more than 6 hours of operation and so facilitates transfer of patients after establishment on PCA in recovery and subsequent mobilisation on the ward. The Graseby PCAS is smaller and lighter than previously available units; it is easier to transport but there is no way of locking to the drip stand. Hopefully, in this country, theft of the unit to obtain narcotics will not be a problem. The patient is well protected from the possibility of third-party interference by the cage locking the syringe to the unit and no boli of fluid or reprogramming can be initiated without the master key.

It is important to note that the batteries of the Graseby must not be allowed to discharge completely; this is unlikely but possible in clinical practice.

It has been previously recommended that PCA pumps should utilise a double push to initiate a demand and to avoid the risk of accidental triggering during movement of the patient.<sup>4</sup> The Graseby button design represents a new and novel approach to this problem. The handset uses pneumatic triggering of a switch housed in the pump. The large button is flush with the top of the handset and requires a definite single push to initiate the dosage cycle. This appears to be a safe design; we have experienced no problems with accidental dosing. It is worth noting, however, that two cases of self-triggering from faulty Graseby handsets have been reported elsewhere,<sup>7,8</sup> although the handset has now been modified to overcome this problem.

The green light present on the 'run' button glows constantly when the unit is ready to provide the patient with a bolus. During the first year of the Acute Pain Service it was noted that this in fact can provide a useful feedback mechanism for patients. If a patient wishes to take several doses before a particularly painful procedure such as physiotherapy or moving out of bed, then this light allows them to demand their dose reliably when the pump is ready. We feel that many patients have benefited from this advice.

The PCA pumps have proven popular with anaesthetic,

surgical and nursing colleagues. The decreasing costs and increased availability of units such as these makes them more attractive to use in the provision of postoperative pain relief rather than purely as a research tool.

#### Acknowledgments

The authors thank Graseby Medical Ltd for loaning the Graseby PCAS. We also thank Professor C. J. Hull for his initial guidance at the start of this study.

#### References

1. SECHZER PH. Objective measurement of pain. *Anesthesiology* 1968; **29**: 209–10.
2. EVANS JM, MACCARTHY JP, ROSEN M, HOGG MIJ. Apparatus for patient-controlled administration of intravenous narcotics during labour. *Lancet* 1976; **1**: 17–18.
3. ROSEN M. Patient controlled analgesia. *British Medical Journal* 1984; **289**: 640–41.
4. HULL CJ. An on-demand analgesia computer (ODAC). In: *Patient-controlled analgesia*. Oxford: Blackwell Scientific Publications, 1985: 83–6.
5. OWEN H, GALVIN RJ, REEKIE RM, TREW AS. Patient-controlled analgesia. Experience of two new machines. *Anaesthesia* 1986; **41**: 1230–5.
6. British Standards Institution. British Standard 5724. Specification for safety of medical electrical equipment. Part 1. General requirements, London: BSI, 1979.
7. HANNING CD, OGDEN B, PARRY P. Graseby PCAS pumps — a potential hazard. *Anaesthesia* 1987; **42**: 559–60.
8. NOTCUTT WG, MORGAN RJM. Introducing patient-controlled analgesia for postoperative pain control into a district general hospital. *Anaesthesia* 1990; **45**: 401–6.



## Occupational health and pollution from anaesthetics

A report of a seminar

M. J. HALSEY

### Summary

*The Control of Substances Hazardous to Health Regulations require employers in the United Kingdom to evaluate and control the risks to health for all their employees from exposure to hazardous substances at work. This applies to those working in hospital operating theatres who may be exposed to anaesthetic gas pollution. These legal requirements coupled with continuing concern about the effective localised control of anaesthetic pollution, the potential chemical interactions in the upper atmosphere, as well as the analysis of the prospective study in the United Kingdom on the health of women doctors, have prompted a reassessment of the topic. Some of the original fears are without foundation but the overall conclusion is that we can still not regard anaesthetic pollution as a problem solved.*

### Key words

*Anaesthetics, gases; trace concentrations.*

*Anaesthetics, volatile; trace concentrations.*

Anaesthetic pollution was a major topic of interest in the 1970s and early 1980s. The original and rather alarming reports prompted a considerable amount of multidisciplinary research ranging from epidemiology, teratology and toxicology to monitoring and control technology. The number of papers on the subject burgeoned<sup>1,2</sup> and even monographs<sup>3</sup> were published. However, over the last 7 years the numbers of publications have waned and many anaesthetists have regarded the problem as 'nonexistent', 'solved' or at least 'put into perspective'.

However, this does not mean that anaesthetic pollution has 'gone away' and a number of coincidental factors have brought the matter back into prominence. These were highlighted at a discussion seminar at Bedford Square on Aspects of Anaesthetic Pollution which took place in February 1990. Invited speakers and discussants included anaesthetists, industrialists, medical physicists and pharmacists as well as senior staff of the Health and Safety Executive.

The seminar opened with Dr M.J. Halsey (*Medical Research Council*) who summarised the *underlying reasons* for continuing concern about the problem. Firstly, careful and systematic monitoring studies indicate that individual trends in anaesthetic pollution levels are not as low as was confidently predicted 10 years ago, at the time at which scavenging devices became commonplace. Second, there are now clear legal requirements in Britain for assessment

of the problem under the Control of Substances Hazardous to Health (COSHH) Regulations which became mandatory in January 1990. However, a balanced overview on the subject must also take into account other factors, including the conclusions of the prospective study in the United Kingdom on the health of women doctors, the issues of environmental pollution on a global scale and the continuing emphasis that no solution can compromise patient safety. All these topics were covered during the meeting by presentations from the leading workers and by wide ranging discussion.

Many anaesthetic departments do not have their working areas monitored for anaesthetic pollution in the belief that scavenging devices have solved the problem. This belief was challenged by two independent investigators who reported their findings from different parts of the United Kingdom. First, Dr J. O'Sullivan (*Principal Pharmacist, Barnsley District Hospital*) discussed the development of methods for determining *personal exposure levels* using diffusive sampling tubes. The importance of personal rather than general sampling was emphasised by the infrared imaging of localised clouds of vapours in operating rooms. The results from Dr O'Sullivan's assessments indicated that there was still a wide range of anaesthetic concentrations in operating theatres, in some cases up to 1000 ppm of nitrous oxide. Much of Dr O'Sullivan's work was carried out in collaboration with Sir Basil

---

M.J. Halsey, MA, DPhil, Head of Research Group, Division of Anaesthesia, Clinical Research Centre, Watford Road, Harrow HA1 3UJ, Middx.

Accepted 21 August 1990.

Houldsworth, who was a pioneer in the field and who continued to take an active interest in the new developments up to his sudden death a few months after the meeting.

The practical levels of anaesthetic pollution have also been extensively studied by Dr W.R. Gray (*University of Glasgow*). He presented a survey of occupational exposure to nitrous oxide in five Glasgow hospitals, which was an update on his earlier reports.<sup>4</sup> The pollution concentrations were in general surprisingly high with the anaesthetists' exposure being greater than 100 ppm nitrous oxide on more than half the occasions. There was evidence that a large part of the exposure could occur in the anaesthetic room. Dr Gray also described the results of surveys of midwives' exposure to nitrous oxide in five hospitals. The mean exposure was over 300 ppm nitrous oxide in two hospitals. The exposures were reduced by use of a scavenging system and by the installation of extractor fans in the delivery room windows. The exposures were still unexpectedly high in the latter case. The general conclusions from these data were that hospital staff do not always use available antipollution facilities and that regular monitoring of staff exposures is required to check the effectiveness of antipollution measures.

Statutory aspects of the Control of Anaesthetic Pollution in the Workplace were considered by Mr A. Simms (*H.M. Principal Inspector of Factories, Health and Safety Executive*). He stated that although evidence of damage to health from waste anaesthetic gases at the workplace is ambiguous, the need for an assessment under the *COSHH Regulations* is clear. An earlier Department of Health Circular<sup>5</sup> indicated that scavenging systems should be fitted to all operating theatres and that exposure to anaesthetic gases should be reduced as far as is practically possible. The same circular also contains advice from the Association of Anaesthetists to their members on safe working practices. These documents are being reviewed and a new guidance note should emerge soon which will include a consideration of other environments (e.g. ambulances and areas where cryosurgery is being practised). The identification of anaesthetic gases and vapours as 'substances hazardous to health' meant that a proper COSHH assessment would have to be made in writing, together with an agreement on policies covering matters such as protection, monitoring, health surveys and training. Subsequent correspondence with the Association of Anaesthetists indicated that the responsibilities for assessments and policies lay with individual hospital management, but it is clear that senior anaesthetists will also have to be involved. There is already some guidance published for the initial assessments in hospitals<sup>6</sup> as well as explanatory booklets on COSHH regulations.<sup>7</sup> There was inevitably some discussion about whether or not inhalation anaesthetics should be covered by these regulations but it was clear that the Health and Safety Executive had already decided that anaesthetics must be included among substances harmful to health. However, the options in relation to target concentrations<sup>8,9</sup> were still under consideration and discussions would soon be taking place with interested parties. The meeting noted the role of the European Commission and the fact that some European countries had already established environmental exposure standards for anaesthetic gases.

Dr R. Knill-Jones (*Acting Head of the Department of*

*Community Medicine, University of Glasgow*) presented some of the hard data from the 10-year prospective study in the UK, supported by the Medical Research Council and endorsed by the Department of Health. The results are based on an annual mailing of a questionnaire to all women doctors aged 40 or less at the time of recruitment and who work in NHS hospitals irrespective of specialty.<sup>10</sup> The final response rate was 89.9% which is generally regarded as satisfactory for exercises of this type. There were significant correlations between miscarriage rates and previous outcome of pregnancy, maternal age, maternal smoking and alcohol consumption. However, there were no significant correlations between miscarriage rates and medical specialty, hours worked in the operating room, or the use (or non-use) of scavenging devices. Similar analyses of congenital abnormality rates yielded negative results. The initial analyses of those children born to anaesthetists indicated that they had 2% lower birth weights which initially appeared to be statistically significant and related to the hours worked in theatres. However, a more detailed analysis has revealed the confounding variable that there are different proportions of Asian doctors in the different specialties in the UK and that children born to Asian women inherently have lower birth weights. A study of this size, which is unique in the field, has yielded a vast amount of data which will continue to take time to analyse fully. However, the overall conclusions are that the original fears of the dangers of exposure to anaesthetic pollution during pregnancy are without foundation.

The wider environmental issues were addressed by Dr M. Logan (*University of Edinburgh*). The issue was whether or not the anaesthetic gases and vapours might contribute to the 'greenhouse effect' or to the depletion of the ozone layer.<sup>11</sup> The present state of knowledge suggests that halothane, enflurane and isoflurane present only a minimal threat to the ozone layer. Nitrous oxide is known to have an ozone depleting potential and is also considered to be a 'greenhouse gas'. However, its interactions are complicated and the final effects could be positive or negative depending on climatic conditions. Dr Logan's own conclusions<sup>12</sup> are supported by those of other investigators<sup>13</sup> and anaesthetic pollution does not appear to have significant environmental consequences.

The obvious immediate solutions to the potential problems of anaesthetic pollution have always been related to anaesthetic equipment. It was therefore particularly appropriate that Mr L. Smith and Mr D. Kent (*Ohmeda, BOC Health Care*) should discuss pollution-free apparatus in the year 2000. They commented that the perceived need for anaesthesia gas scavenging equipment varies considerably from clinician to clinician and from country to country. Antipollution equipment is not particularly exciting, solely as a business opportunity, partly because of this fact. However, manufacturers of anaesthesia systems whose promotional platform is highly focused on patient/user safety will continue to develop scavenging technology because of the 'complete' systems philosophy.

Britain has possibly the most stringent performance requirements for the scavenging equipment<sup>14</sup> being designed today. Patient safety is the key issue. Health and safety issues are likely to become important as well, with acceptance that changes in procedure can produce dramatic reductions in the levels of personal exposure, for example leak-free connexions or intravenous anaesthesia. The cost

of anaesthesia may lead in the future to the development of intelligent scavenging systems to minimise wastage of agent and these may be tailored to suit the differing needs of open and closed systems.

The meeting as a whole clearly demonstrated that the problem of anaesthetic pollution is not going to go away and although some aspects are now satisfactorily answered, new questions were raised. The immediate issues are related to the implementation and consequences of COSHH. These include agreement on acceptable atmospheric levels of nitrous oxide and volatile agents, approval of monitoring procedures both in terms of techniques and frequency as well as a greater understanding of the relationships between peak and mean levels and the effect of exposure time.<sup>1,15</sup> The financial implications will be obvious to every new Director of Anaesthesia/Theatre Services because the major stumbling block to the solutions is the fact that Safety costs . . .

### References

1. DAVENPORT HT, HALSEY MJ, WARDLEY-SMITH B, WRIGHT BM. Measurement and reduction of occupational exposure to inhaled anaesthetics. *British Medical Journal* 1976; **2**: 1219-21.
2. REDFERN N. Morbidity among anaesthetists. *British Journal of Hospital Medicine* 1990; **43**: 377-81.
3. COHEN EN. *Anesthetic exposure in the workplace*. Littleton, Massachusetts: P.S.G. Publishing, 1980.
4. GRAY WM. Occupational exposure to nitrous oxide in four hospitals. *Anaesthesia* 1989; **44**: 511-4.
5. Department of Health. Pollution of Operating Departments etc. by Anaesthetic Gases. *Health Circular HC (76) 38*. London: Department of Health, 1976.
6. GLASS DC, HALL AJ, HARRINGTON JM. *The control of substances hazardous to health: guidance for the initial assessment of hospitals*. London: Her Majesty's Stationery Office, 1989: 108-13.
7. Health and Safety Executive. *COSHH assessments: a step by step guide to assessment and the skills needed for it*. London: Her Majesty's Stationery Office, 1989.
8. HALSEY MJ. Maximum safe levels of anaesthetic contamination in operating rooms. *British Journal of Anaesthesia* 1978; **50**: 633P.
9. National Institute for Occupational Safety and Health. *Criteria for a recommended standard for occupational exposure to waste anesthetic gases and vapors*. DHEW (NIOSH) Publication No. 77-140. Washington: Department of Health, Education and Welfare, 1977.
10. SPENCE AA, WALL RA, NUNN JF. Environmental safety of the anaesthetist. In: Nunn JF, Utting JE, Brown BR, eds. *General anaesthesia*, 5th edn. London: Butterworths, 1989: 595-608.
11. BROWN AC, CANOSA-MAS CE, PARR AD, WAYNE RP, PIERCE JMT. Halothane and the atmosphere. *Lancet* 1989; **2**: 279.
12. LOGAN M, FARMER JG. Anaesthesia and the ozone layer. *British Journal of Anaesthesia* 1989; **63**: 645-7.
13. BROWN AC, CANOSA-MAS CE, PARR AD, PIERCE JMT, WAYNE RP. Tropospheric lifetimes of halogenated anaesthetics. *Nature* 1989; **341**: 635-7.
14. *British Standards*, Specifications for Active Anaesthetic Scavenging Systems, BS 6834. London: British Standards Institution, 1987.
15. GARDNER RJ. Inhalation anaesthetics—exposure and control: a statistical comparison of personal exposures in operating theatres with and without gas scavenging. *Annals of Occupational Hygiene* 1989; **33**: 159-73.

## Forum

### The laryngeal mask airway in children A fibreoptic assessment of positioning

S. J. Rowbottom, FFARCS, Senior Registrar, D. L. Simpson, FFARCS, D. Grubb, FFARCS, Consultants, Department of Anaesthetics, Royal Hospital for Sick Children, Sciennes Road, Edinburgh, Scotland, UK.

#### Summary

*Clinical and fibreoptic assessment of the positioning of the laryngeal mask airway was performed in 100 children. Clinical observation indicated a patent airway in 98% and severe airway obstruction in 2% of cases. Perfect positioning, as judged by fibreoptic laryngoscopy, was found in 49% and the epiglottis was within the mask in 49%. Fibreoptic evidence of partial airway obstruction in 17% was not detected clinically.*

#### Key words

*Anaesthesia; paediatric.*

*Equipment; laryngeal mask airway, fibreoptic laryngoscope.*

The laryngeal mask airway (LMA) was designed following studies on the cadaveric adult larynx.<sup>1</sup> Three out of the four sizes of laryngeal mask currently available are appropriate for use in infants and children; size 1 in infants up to 6.5 kg, size 2 for infants and children from 6.5 to 25 kg and size 3 for larger children or small adults. Sizes 1 and 2 are scaled-down versions of adult masks. However, the relative anatomy of the larynx of infants and young children is known to differ from that of the adult; a higher and more anteriorly placed glottis with a relatively large floppy epiglottis may make correct placement of the LMA more difficult. We assessed the position of three sizes of LMA in anaesthetised children using a fibreoptic laryngoscope.

#### Method

Observations were made on 100 consecutive healthy children (inpatients and outpatients) presenting for elective general surgery. Patients who were not fasted or those requiring tracheal intubation or intermittent positive pressure ventilation as part of the anaesthetic technique were not studied. Masks were inserted by the authors; two had previous experience with the technique in children and one had experience in adults. Clinical assessment of airway patency and fibreoptic laryngoscopy were performed by two observers.

Premedication and the method of induction of anaesthesia (inhalational with halothane in nitrous oxide and oxygen or intravenous with propofol 3 mg/kg) were at the discretion of the anaesthetist. Masks were all inserted when the children were anaesthetised and breathing 4% halothane in nitrous oxide and oxygen (2:1). A Bain

anaesthetic system was used in children over 15 kg and an Ayre's T-piece in smaller children.

The LMA was inserted in a standard fashion according to the maker's recommendations (Intavent Laryngeal Mask Airway, DJ. Colgate Ltd., UK) and the cuff inflated with air. Correct positioning of the LMA was clinically determined as follows; resistance to continued advancement of the LMA with forward bulging of the larynx during insertion and outward movement of the LMA with cuff inflation. Airway patency was confirmed by observing synchronous respiratory movements of the chest and anaesthetic reservoir bag, by a lack of indrawing of intercostal and supraclavicular spaces and by confirming, on auscultation, air entry in both axillae when gently inflating the lungs.

The actual position of the LMA was then ascertained by fibreoptic laryngoscopy (Olympus ENF P2) while an assistant prevented the mask from moving. Laryngoscopic findings were classified as shown in Table 1. According to the findings the LMA was either taped in position or reinserted and the laryngoscopy repeated. A maximum of three insertions were to be permitted. If positioning was still unsatisfactory the use of the LMA would be abandoned and an

Table 1. Laryngoscopic findings.

Group 1, larynx only seen.
Group 2, epiglottis and larynx seen.
Group 3, epiglottis impinging on grille, larynx seen.
Group 4, kinked laryngeal mask airway.
Group 5, epiglottis downfolded, larynx not seen.

Accepted 8 October 1990.

**Table 2.** Fibreoptic laryngoscopic findings for each mask size after initial insertion.

Mask size	1	2	3
Group 1	3	36	10
Group 2	3	14	13
Group 3	1	10	4
Group 4	0	2	0
Group 5	0	4	0
Total	7	66	27

oropharyngeal airway used instead. The conduct of the remainder of the anaesthetic was left to the discretion of the individual anaesthetist.

The electrocardiogram (ECG) and arterial oxygen saturation (Nellcor N 100 pulse oximeter) were monitored continuously from induction and for 3 minutes after insertion of the LMA. Printed recordings of heart rate and oxygen saturation were obtained every 30 seconds; the lowest saturation and the time at which it occurred were noted.

### Results

There were 85 male and 15 female children. Their mean age was 5.6 years (range 0.11–15.5 years) and mean weight 21.6 kg (range 5–65 kg). One hundred masks were used for the first insertion: seven size 1, 66 size 2 and 27 size 3. The body weight for age of each patient was analysed by reference to standard growth charts. Three children were above the 97th centile (two size 2 mask and one size 3 mask) and only one was below the 3rd centile (size 2 mask).

A clinically patent airway was observed in 98% of cases and clinically severe airway obstruction was present in 2% after the first insertion of the LMA.

Table 2 shows details of the fibreoptic assessment of positioning of the LMA. In 49% of cases the larynx alone was seen and positioning was perfect. The larynx was directly seen in 79% (groups 1 and 2). The epiglottis impinged on the grille of the LMA in 15% but the larynx could be visualised by manipulation of the fibreoptic laryngoscope. Overall, the larynx was visible in 94% of cases.

Two size 2 LMA were partially kinked. Partial airway obstruction based on fibreoptic findings was thus present in 17% (groups 3 and 4). Kinked tubes were reinserted uneventfully. Downfolding of the epiglottis was observed in four cases and caused severe airway obstruction on clinical assessment in two of these. Downfolding of the epiglottis was relieved by substituting a size 1 for a size 2 LMA in two infants (both weighing 7 kg) and by reinsertion of the size 2 LMA in two infants of 11 kg.

The pulse oximetry data were examined in relation to the laryngoscopic findings (Table 3). After insertion of the LMA the minimum arterial oxygen saturation at any time, in any patient, was 88% and arterial desaturation below a value of 90% occurred in only three patients. Pulse

**Table 3.** Oximetry data in relation to laryngoscopic findings.

	Laryngoscopic group				
	1	2	3	4	5
Number of patients with $\text{SaO}_2 < 90\%$	2	1	0	0	0
Minimum $\text{SaO}_2$	89	88	93	95	93

$\text{SaO}_2$ , arterial oxygen saturation.

oximetry was not a reliable guide to the laryngoscopic findings.

### Discussion

Accounts of the development of the laryngeal mask airway describe the position of the correctly placed LMA as surrounding the larynx with the epiglottis lying outside the cuff.<sup>1,2</sup> The manufacturers' instructions for usage depict this situation. Our observations with the fibreoptic laryngoscope showed that perfect positioning was found in only 49% of cases overall, and that the epiglottis was inside the LMA, in the grille or downfolded, in 49%. These observations were not the result of an abnormal distribution of weight for age because only four children were outside the 3rd to 97th growth centiles.

Severe airway obstruction attributable to the LMA was clinically obvious. Substitution of a size 1 LMA for the original size 2 mask relieved the obstruction in the two infants weighing 7 kg. We also found the size 2 LMA rather bulky to insert in infants around this weight. There were two cases with a downfolded epiglottis and two kinked LMAs which were only recognised by fibreoptic laryngoscopy. When downfolding of the epiglottis occurred the epiglottis was relatively long and thin and was closely apposed to the posterior pharyngeal wall, making it difficult to position the LMA properly. Pulse oximetry remained normal in all cases and the epiglottis was seen to move a little with respiratory effect thus permitting some gas exchange around it.

The appearance of the epiglottis in the grille of the LMA constitutes endoscopic evidence of partial airway obstruction (group 3). However, as the larynx could be directly visualised by manipulating the fibreoptic laryngoscope but not the epiglottis, we accepted this as an unobstructed upper airway and decided to leave these masks in place. There was no clinical evidence of airway obstruction in these patients. Presumably, some encroachment by the epiglottis into the mask or grille of the LMA can be tolerated, since the 'tubes' of the LMA and larynx are deliberately joined end-on (as opposed to one inside the other with a tracheal tube) to give a wider lumen.

In reports of experience with the LMA in adults and children the incidence of a clear airway, as judged by clinical criteria, was 96–98%, with few major problems occurring.<sup>3,4</sup> Our clinical findings are comparable; a clear airway in 98% and severe obstruction in 2%. Fibreoptic laryngoscopy in 50 adults demonstrated complete airway obstruction in 1% and partial obstruction in 10% of cases.<sup>5</sup> In our study with children the incidences were 2% and 19%, respectively.

Use of the LMA in routine paediatric anaesthetic practice is increasing and its use in the management of the difficult airway has been described, albeit with some problems and reservations.<sup>4,6–8</sup> In adults the LMA has been used to aid blind, guided intubation techniques (using gum elastic bougies or tracheal tube passed down the LMA).<sup>9–11</sup> Our observations suggest caution in the use of the LMA as an aid to this technique in children; in 51% of our cases there might have been some impediment to blind passage of a bougie or introducer.

Fibreoptic assessment of the LMA in children shows that while positioning may not be anatomically perfect, its function is clinically satisfactory in the majority of children.

### Acknowledgments

The authors thank Mr W.H. Bissett, Mr G.A. MacKinlay and theatre staff at the Royal Hospital for Sick Children, Edinburgh for their help during the study.

## References

1. BRAIN AIJ. The laryngeal mask: a new concept in airway management. *British Journal of Anaesthesia* 1983; **55**: 801–5.
2. BRAIN AIJ, MCGHEE TD, MCATEER EJ, THOMAS A, ABU-SAAD MAW, BUSHMAN JA. The laryngeal mask airway: development and preliminary trials of a new type of airway. *Anaesthesia* 1985; **40**: 356–61.
3. BRODRICK PM, WEBSTER NR, NUNN JF. The laryngeal mask airway: a study of 100 patients during spontaneous breathing. *Anaesthesia* 1989; **44**: 238–41.
4. MASON DG, BINGHAM RM. The laryngeal mask airway in children. *Anaesthesia* 1990; **45**: 760–3.
5. PAYNE J. The use of the fiberoptic laryngoscope to confirm the position of the laryngeal mask. *Anaesthesia* 1989; **44**: 865.
6. GREBENIK CR, FERGUSON C, WHITE A. The laryngeal mask airway in pediatric radiotherapy. *Anesthesiology* 1990; **72**: 474–7.
7. BEVERIDGE ME. Laryngeal mask anaesthesia for repair of cleft palate. *Anaesthesia* 1989; **44**: 656–7.
8. RAVALIA A, GODDARD JM. The laryngeal mask and difficult tracheal intubation. *Anaesthesia* 1990; **45**: 168.
9. CHADD GD, ACKERS JW, BAILEY PM. Difficult intubation aided by the laryngeal mask airway. *Anaesthesia* 1989; **44**: 1015.
10. ALLISON A, MCCRORY J. Tracheal placement of a gum elastic bougie using the laryngeal mask airway. *Anaesthesia* 1990; **45**: 419–20.
11. BRAIN AIJ. Three cases of difficult intubation overcome by the laryngeal mask airway. *Anaesthesia* 1985; **40**: 353–5.

*Anaesthesia*, 1991, Volume 46, pages 491–493

### Visual evoked potentials and visual acuity after transurethral resection of the prostate

S. Mantha, MD, Assistant Professor, S. M. Rao, MD, DA, FCCP, Professor and Head of Department, A. K. Singh, MD, DM(Neuro), Assistant Professor of Neurology, S. Mohandas, MD, DM(Neuro), Professor of Neurology, B. Surya Prakash Rao, MS, MCh(Urol), Assistant Professor of Urology, N. Joshi, MSc, PhD, Lecturer, Department of Gastroenterology, Nizam's Institute of Medical Sciences, Hyderabad 500 482, India.

#### Summary

Changes in visual evoked potentials, visual acuity, blood ammonia levels and serum electrolytes ( $\text{Na}^+$  and  $\text{K}^+$ ) after transurethral resection of the prostate using glycine as an irrigating fluid performed under subarachnoid block were studied in 12 patients, in the pre-operative and immediate postoperative periods. Visual evoked potentials ( $P_{100}$  latency), recorded by shift of a checkerboard pattern, increased significantly from a pre-operative value of mean (SEM) 101.18 (1.63) msec in the right eye, and 102.5 (1.47) msec in the left eye to 108.91 (1.8) msec ( $p < 0.01$ ) and 108.08 (2.53) msec ( $p < 0.01$ ) respectively in the postoperative phase. There were no changes in visual acuity as assessed by a Snellen's chart, blood ammonia levels and serum electrolyte concentrations. The amount of glycine used intra-operatively for irrigation ranged from 3 to 31 litres.

#### Key words

Complications; glycine absorption.

Surgery, urological; transurethral resection of the prostate.

Monitoring; visual evoked potentials.

Transurethral resection of the prostate (TURP) syndrome, comprising central nervous system (CNS) and cardiovascular disturbances, has been recognised for many years.<sup>1–8</sup> These complications, which include prolonged sedation after surgery,<sup>1,2</sup> coma,<sup>3,4</sup> visual disturbances,<sup>5–7</sup> cardiovascular instability and even cardiac arrest<sup>8</sup> are thought to be caused by excessive absorption of irrigating fluid during surgery. Glycine is widely used as an irrigating fluid for TURP. Recently, the cause of altered visual physiology, prolongation of visual evoked potential (VEP) latency, has been attributed to glycine itself<sup>6</sup> or its metabolite.<sup>9</sup>

The present study was designed to evaluate changes in VEPs and visual acuity after TURP using glycine as an irrigating fluid, and to correlate these with changes in concentrations of blood ammonia and serum electrolytes. Although suppression of VEPs after TURP have been reported,<sup>6,9</sup> changes in visual acuity assessed by a Snellen's chart have not been studied before, to our knowledge.

#### Methods

The protocol for the study was approved by the ethics committee at Nizam's Institute of Medical Sciences, Hyderabad. Thirteen consecutive patients scheduled for elective TURP were studied, after obtaining informed consent. The patients belonged to ASA 1 and 2.

The patients were not premedicated. Pre-operative VEPs and visual acuity were determined approximately 1–2 hours before surgery. VEPs were recorded using the Neuromatic 2000 c (Dantec) electromyographic and evoked potential system. Active and reference electrodes were placed at  $\text{O}_z$  and  $\text{C}_z$  positions respectively with the ground electrode strapped around the wrist. The visual stimulus used was a checkerboard (check size 15 mm) pattern reversal at 2 Hz which was placed at a distance of 1.5 m from the patient. The squares reversed colour (black/white) without changing the total light output (luminance). Filter settings

used were 0.5 Hz (lower) and 1 kHz (upper). At least 200 responses were averaged (using the autorejection mode) to form the VEP. The recording was repeated to see the reproducibility of VEP waveforms, and only consistently reproducible potentials were accepted for analysis. The latency of  $P_{100}$  was measured electronically by placing the cursor over the tip of the  $P_{100}$  peak. The amplitude of  $P_{100}$  was also noted. Each eye was studied separately by patching the other. Visual acuity was studied using the Snellen's chart placed at a distance of 6 m from the patient. Patients who wore spectacles continued to wear them while VEPs and visual acuity were studied.

A 16-G cannula was placed in a peripheral vein under local anaesthesia, and samples taken for estimating blood ammonia and serum electrolyte concentrations. An infusion of lactated Ringer's solution was started via the peripheral vein. Subarachnoid block with 5% lignocaine was performed under aseptic conditions at the  $L_{3-4}$  interspace, with the patients in the sitting position. They continued in that position for the next 10 minutes to obtain a saddle block. Glycine 1.5% was used as the irrigating fluid during the operation. Monitoring during surgery was by continuous ECG and automated noninvasive blood pressure by Datex Cardiocap. Blood loss was estimated subjectively by the surgeon. Resection time and amount of glycine used for irrigation were noted.

A venous blood sample was obtained to estimate serum electrolytes and blood ammonia concentrations at the end of surgery. Postoperative VEPs and visual acuity were studied within an hour after surgery. Blood ammonia was estimated by the ultraviolet enzymatic method using the commercial kit supplied by Boehringer-Mannheim. Normal values were considered to be 25–94  $\mu\text{g/dlitre}$  for men.

Data were analysed using the paired *t*-test and coefficients of correlation ( $\rho$ ) for comparing the variables wherever appropriate. Statistical significance was considered at  $p < 0.05$ . All data are presented as mean (SEM).

### Results

One patient with bilateral cataract in whom pre-operative VEPs could not be obtained, was excluded from the study. In another patient VEPs could not be obtained from a blind right eye. These factors resulted in the analysis of VEPs and visual acuity from 23 eyes in 12 patients. The patients' ages ranged from 60 to 83 years with a mean of 68.28 (2.29).

Resection time ranged from 30–120 minutes with a mean of 53.75 (7.27) minutes during which 14.38 (2.59) litres (range 3–31) of 1.5% glycine was required for irrigation. During operation, all patients were haemodynamically stable and had no alteration in mental status. Blood loss as assessed subjectively by the surgeon, ranged from 150 to 300 ml. Infusion of lactated Ringer's solution during the surgery ranged from 500–1000 ml. No patient required blood transfusion.

There was a statistically significant prolongation of the VEP latency and suppression of amplitude after surgery (Table 1). There were no changes in visual acuity after

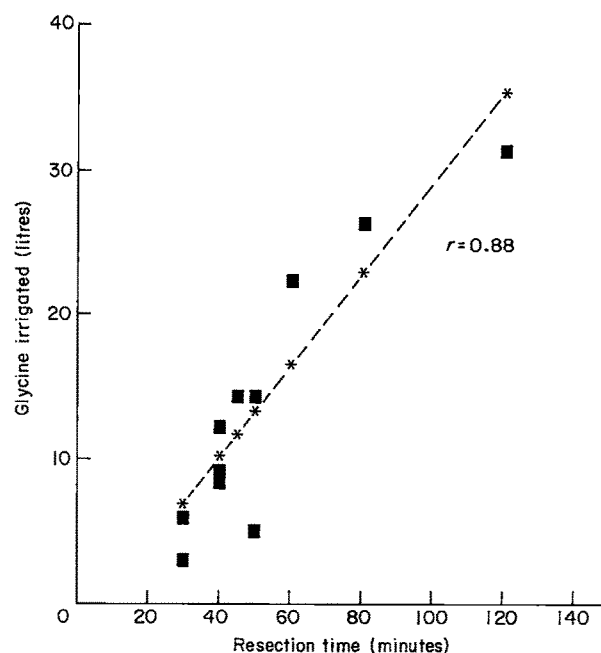


Fig. 1. Correlation between resection time and amount of glycine irrigated.

operation in 11 patients, while a decrease from 6/9 (both eyes) to 6/12 was observed in one patient. This patient demonstrated an increase in the VEP latency from 100 msec (right eye) and 98 msec (left eye) to 102 msec and 104

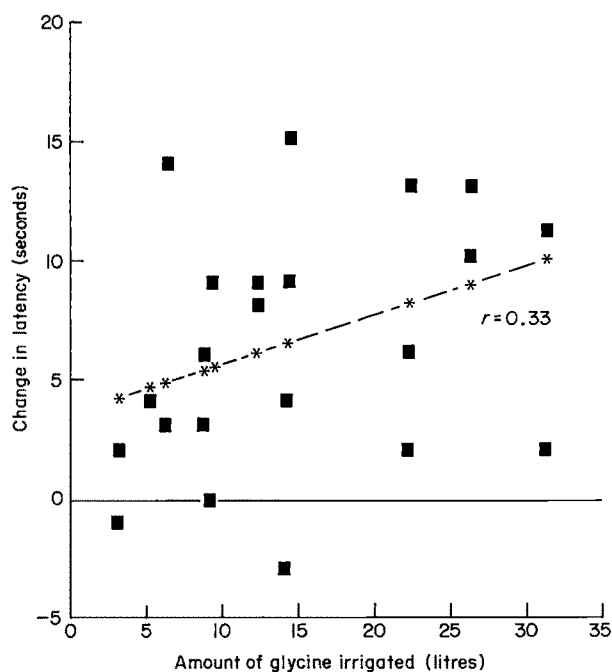


Fig. 2. Correlation between amount of glycine irrigated and change in latency.

Table 1. Changes in VEPs after TURP, mean (SEM).

VEP	Before operation	After operation	p value
Latency of right eye; msec	101.18 (1.63)	108.91 (1.80)	<0.01
Latency of left eye; msec	102.50 (1.47)	108.08 (2.53)	<0.01
Amplitude of right eye; $\mu\text{V}$	8.22 (0.89)	5.37 (0.78)	<0.05
Amplitude of left eye; $\mu\text{V}$	8.10 (1.12)	5.31 (0.59)	<0.01



**Table 2.** Changes in blood ammonia and serum electrolyte concentration, mean (SEM).

	Before operation	After operation	p value
Blood ammonia; $\mu\text{g/dlitre}$	57.15 (13.38)	58.88 (12.39)	ns
Serum Na; mmol/litre	142.33 (1.55)	141.33 (1.90)	ns
Serum K; mmol/litre	4.46 (0.13)	4.09 (0.14)	ns

ns, not significant.

msec respectively. There was a strong correlation between the resection time and the amount of glycine irrigant ( $r = 0.88$ ,  $p < 0.01$ , Fig. 1). No such relationship could be found between the latter and the change in VEP latency ( $r = 0.33$ , Fig. 2). There were no changes in blood ammonia and serum electrolyte concentrations (Table 2).

### Discussion

TURP syndrome results from excessive absorption of the irrigating fluid via the open prostatic venous sinuses during resection. Visual disturbances<sup>5</sup> including total blindness<sup>7</sup> and prolongation of VEP latency<sup>6,9</sup> following TURP with glycine irrigation have been well documented. These aberrations in the visual physiology are thought to be caused by the action of glycine as an inhibitory neurotransmitter in the CNS and retina.<sup>10</sup> The role of glycine in suppressing the VEP responses is also demonstrated by experimental studies.<sup>11,12</sup>

VEPs which reflect the functional integrity of the retina and the optic tracts provide an objective and a highly sensitive method of analysing their abnormalities. Pattern shift and luminance change are the two commonly used stimuli for recording VEPs. The former was preferred in this study because of its higher sensitivity and lower variability.<sup>13</sup> Objective assessment of visual acuity was found to be lacking in the available literature. Blood ammonia and serum electrolyte concentration were estimated because hyperammonaemia<sup>2</sup> and dilutional hyponatraemia<sup>3</sup> have been implicated in the pathogenesis of the TURP syndrome.

Postoperative suppression of VEPs demonstrated in this study correlate well with the results of the two clinical studies reported earlier.<sup>6,9</sup> Glycine absorbed into the systemic circulation is probably responsible for alterations in the VEPs. The amount absorbed is determined by the duration of resection, quantity of the irrigant fluid used, number of open prostatic venous sinuses and the amount of the fibrous tissue in the gland. The influence of these last two factors may account for the lack of correlation between the amount of glycine irrigant and the change in VEP latency in our observation. Dramatic elevation of glycine level is often associated with normal levels of ammonia following TURP with glycine irrigation.<sup>2,6</sup> It may be inferred that our patients could have had an elevated systemic glycine concentration because there were no changes in the blood ammonia levels. Glycine itself and/or other metabolites other than ammonia may be responsible for the changes in visual electrophysiology observed in our study.

No changes occurred in the visual acuity of our patients despite prolonged VEP latency, as was shown in an earlier study.<sup>9</sup> In a recent study by Wang *et al.*,<sup>6</sup> subjective visual disturbances correlated better with serum glycine levels than with the increase in VEP latency. These authors considered that retinal condition and cerebral cortical

condition are affected by different mechanisms in the TURP syndrome.

We have demonstrated significant prolongation of P<sub>100</sub> latency in the immediate postoperative period following TURP surgery with glycine irrigation. There were no subjective changes in vision. No alterations in blood ammonia or serum electrolyte concentrations were observed.

### Acknowledgments

The authors thank Dr P.V. Rao, Assistant Professor of Medicine, for his help in data analysis. Technical assistance from Mr M.D.K. Rao and Ms S. Sailaja, in recording VEPs is gratefully acknowledged.

### References

1. ROESCH RP, STOELTING RK, LINGEMAN JE, KAHNOSKI RJ, BACKES DJ, GEPHARDT SA. Ammonia toxicity resulting from glycine absorption during a transurethral resection of the prostate. *Anesthesiology* 1983; **58**: 577-9.
2. HOEKSTRA PT, KAHNOSKI R, MCCAMISH MA, BERGEN W, HEETDERKS DR. Transurethral prostatic resection syndrome—a new perspective: encephalopathy with associated hyperammonemia. *Journal of Urology* 1983; **130**: 704-7.
3. HENDERSON DJ, MIDDLETON RG. Coma from hyponatremia following transurethral resection of the prostate. *Urology* 1980; **15**: 267-71.
4. RYDER KW, OLSON JF, KAHNOSKI RJ, KARN RC, OEI TO. Hyperammonemia after transurethral resection of the prostate: a report of 2 cases. *Journal of Urology* 1984; **132**: 995-7.
5. OVASSAPIAN A, JOSHI CW, BRUNNER EA. Visual disturbances: an unusual symptom of transurethral prostatic resection reaction. *Anesthesiology* 1982; **57**: 332-4.
6. WANG JML, CREEL DJ, WONG KC. Transurethral resection of the prostate, serum glycine levels, and ocular evoked potentials. *Anesthesiology* 1989; **70**: 36-41.
7. RUSSEL D. Painless loss of vision after transurethral resection of the prostate. *Anaesthesia* 1990; **45**: 218-21.
8. CHARLTON AJ. Cardiac arrest during transurethral prostatectomy after absorption of 1.5% glycine. A case report and review of the literature. *Anaesthesia* 1980; **35**: 804-5.
9. CASEY WF, HANNON V, CUNNINGHAM A, HEANEY J. Visual evoked potentials and changes in serum glycine concentration during transurethral resection of the prostate. *British Journal of Anaesthesia* 1988; **60**: 525-9.
10. PYCOCK CJ, KERWIN RW. The status of glycine as a supraspinal neurotransmitter. *Life Sciences* 1980; **28**: 2679-86.
11. KOROL S. The effects of glycine on the rabbit retina: averaged ERG and averaged visual evoked responses. *Experientia* 1973; **29**: 984-5.
12. WANG JML, WOMG KC, CREEL DJ, CLARK WM, SHAHANGIAN S. Effects of glycine on hemodynamic responses and visual evoked potentials in the dog. *Anesthesia and Analgesia* 1988; **64**: 1071-7.
13. CHIAPPA KH, ROPPER AH. Evoked potentials in clinical medicine. Part I. *New England Journal of Medicine* 1982; **306**: 1140-9.

### End-tidal CO<sub>2</sub> monitoring in mitral stenosis patients undergoing closed mitral commissurotomy

G. D. Puri, MD, Assistant Professor, R. Venkatraman, MD, Senior Resident, H. Singh, Professor, Post Graduate Institute of Medical Education and Research, H. No. 2095, Sector 15/c, Chandigarh, India 160015.

#### Summary

Arterial to end-tidal carbon dioxide difference ( $P(a-E)CO_2$ ) was recorded in 20 mitral stenosis patients (group A) for closed mitral commissurotomy and 20 healthy individuals (group B) for elective limb surgery. Mitral stenosis patients showed a greater difference than group B patients. Repeated measurements of  $P(a-E)CO_2$  in mitral stenosis patients at various stages of closed mitral commissurotomy not only showed a mean increase from before thoracotomy but there was also no correlation between  $P(a-E)CO_2$  before thoracotomy with that after thoracotomy, after commissurotomy or after chest closure. This indicated that end-tidal CO<sub>2</sub> monitoring was unsuitable to measure adequacy of ventilation during closed mitral commissurotomy.

#### Key words

Heart; mitral valve.

Carbon dioxide; elimination, tension, alveolar.

Continuous measurement of end-tidal carbon dioxide tension  $PE'CO_2$  has been recommended for noninvasive estimation of arterial  $PCO_2$ , but a major concern is that  $PE'CO_2$  may substantially underestimate  $Paco_2$ .<sup>1-3</sup> To overcome this problem it has been suggested that an initial  $P(a-E)CO_2$  be determined and added to all subsequent readings of  $PE'CO_2$ .<sup>4</sup> This presumes stability of  $P(a-E)CO_2$  throughout surgical procedures. Raemer<sup>5</sup> refuted this idea based on repeated measurements of  $P(a-E)CO_2$  in patients for open heart surgery and major abdominal vascular procedures.

Patients with mitral stenosis (MS) have a ventilation perfusion mismatch which may increase  $P(a-E)CO_2$  as compared to normal individuals. We investigated  $P(a-E)CO_2$  in mitral stenosis patients at various stages of closed mitral commissurotomy (CMC) to evaluate the validity of  $PE'CO_2$  in estimating  $Paco_2$ .

#### Material and methods

Twenty mitral stenosis patients (group A) planned for CMC and 20 healthy individuals for peripheral limb surgery (group B) were studied. An informed consent was obtained from all patients for radial artery cannulation and blood sampling. Patients were premedicated with 0.15 mg/kg of morphine and 0.5 mg/kg of promethazine one hour before induction. Anaesthesia was induced with 0.1 mg/kg morphine, 0.12 mg/kg pancuronium bromide and a sleep dose of thiopentone 3-5 mg/kg given slowly. After tracheal intubation patients' lungs were ventilated with 35:65 O<sub>2</sub>:N<sub>2</sub>O mixture by a Servo ventilator 900B. A carbon dioxide analyser 930, which uses an infrared sensor, was

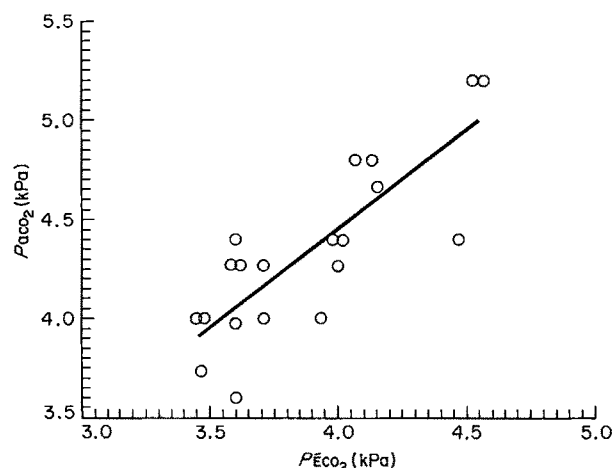
used to detect end-tidal CO<sub>2</sub> concentration. The infrared analyser was regularly calibrated according to manufacturer's specifications. A square wave flow pattern was used with 25% inspiration and 10% inspiratory pause. Patients lungs were ventilated at around 16 breaths per minute with the minute ventilation adjusted to keep end-tidal CO<sub>2</sub> concentration ( $FE'CO_2$ ) around 4%. A radial artery catheter was inserted to allow withdrawal of arterial blood for blood gas analyses. An arterial sample was taken before starting surgery but after at least 10 minutes of stable ventilation after tracheal intubation. End-tidal carbon dioxide concentration was also recorded at that time.

The study was extended in MS (group A) patients to beyond the pre-incision (stage I) stage. Arterial blood samples were taken in these patients after thoracotomy (stage II), after valvotomy (stage III) and after chest closure (stage IV).  $FE'CO_2$  was also noted at the time of sampling. Only patients with stable haemodynamics at the time of sampling were studied.

Arterial blood gas samples were analysed with a radiometer ABL2 and values were corrected to patients' body temperature.<sup>6</sup> Values of  $FE'CO_2$  were corrected for variation of humidity, presence of N<sub>2</sub>O and barometric pressure.<sup>7</sup> Coefficients of variation of carbon dioxide measurements were less than 0.01. Statistical analyses of results were made using Student's *t*-test and linear regression.  $P(a-E)CO_2$  values before start of surgery were compared between the two groups by unpaired *t*-test. A correlation was attempted between  $Paco_2$  and  $PE'CO_2$  in both groups. When repeated determinations were made at different stages of surgery in mitral stenosis patients a correlation coefficient was determined between initial  $P(a-E)CO_2$  (stage I) and subsequent  $P(a-E)CO_2$  (i.e. stages II, III and IV).

Table 1. Patient data. Values are expressed as mean (SD).

	Group A		Group B		p
Age; years	32.5	(6)	30.8	(7.5)	> 0.05
Weight; kg	49.5	(8)	51.6	(5.8)	> 0.05
Paco <sub>2</sub> ; mmHg	32.5	(3.2)	32.0	(1.9)	> 0.05
P(a-E')CO <sub>2</sub>	3.51	(1.84)	0.93	(1.55)	< 0.01
Pao <sub>2</sub> ; mmHg	120.8	(26.1)	150.2	(15.3)	< 0.01

Fig. 1. Regression line for  $P_{aCO_2}$  and  $P_{E'}CO_2$  at prethoracotomy stage in group A.

### Results

The two groups were comparable with respect to age, weight and  $P_{aCO_2}$ . Mitral stenosis patients had significantly higher  $P(a-E')CO_2$  and lower  $P_{aO_2}$  as compared to normal individuals (Table 1).  $P_{aCO_2}$  was positively correlated with  $P_{E'}CO_2$  in both groups ( $p < 0.05$ ). Figure 1 depicts the relation between  $P_{aCO_2}$  and  $P_{E'}CO_2$  values at stage I ( $r = 0.81$ ,  $p < 0.01$ ). When repeated determinations were made initial  $P(a-E')CO_2$  did not correlate with subsequent  $P(a-E')CO_2$  during CMC surgery (Table 2). Variation of  $P(a-E')CO_2$  at various stages in all mitral stenosis patients is plotted in Figure 2.

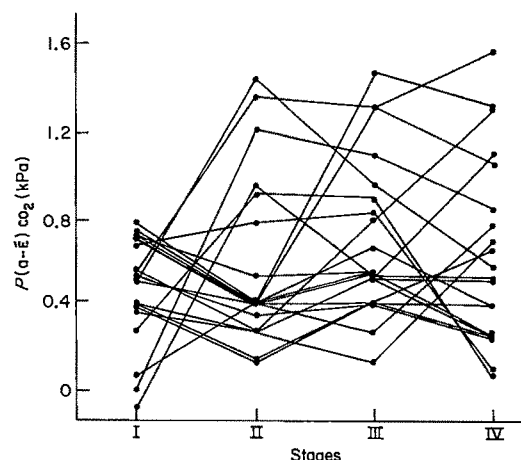
### Discussion

Closed mitral commissurotomy is still a common operation for treatment of patients with mitral stenosis.<sup>8</sup> These patients have decreased lung compliance and increased airway resistance which results in inter- and intraregional nonhomogeneities and maldistribution of inspired air.<sup>9</sup> Pulmonary vascular congestion and oedema can interfere with peripheral airway function<sup>10-14</sup> which results in a relatively greater increase in distal airway resistance. This also causes an increase in closing volume resulting in earlier than normal closure of dependent airways which leads to air trapping and increased residual volume. Supine posture and anaesthesia both result in a decrease in functional residual capacity which may further increase the chances of airway collapse and  $\dot{V}/\dot{Q}$  mismatch.<sup>15</sup> This may be the cause of increased  $P(a-E')CO_2$  and lower  $P_{aO_2}$  in mitral stenosis.

Although mean  $P(a-E')CO_2$  in mitral stenosis patients rose during surgery, the lack of correlation of the initial difference with subsequent differences at various stages of surgery proves that the relationship cannot be predicted. Figure 2 shows that in some patients  $P(a-E')CO_2$  increased while in others it decreased during surgery. This shows that calibration of  $P(a-E')CO_2$  with initial measurements as

Table 2. Mean  $P(a-E')CO_2$  at various stages of CMC surgery, and their correlation with preincision values. (Stage I, preincision; stage II, after thoracotomy; stage III, after valvotomy, stage IV, after chest closure).

Stage	n	$P(a-E')CO_2$ (SD)	r with $P(a-E')CO_2$ of stage I	p
I	20	0.47 (0.25)		
II	20	0.57 (0.41)	0.29	> 0.05
III	20	0.70 (0.38)	0.06	> 0.05
IV	20	0.66 (0.44)	0.06	< 0.05

Fig. 2. Arterial to end-tidal  $CO_2$  difference at various stages of commissurotomy in 20 studied patients.

suggested by Whitesell *et al.*<sup>4</sup> is not reliable in mitral stenosis patients for CMC. An initial difference between arterial and end-tidal  $CO_2$  is not necessarily representative of subsequent differences. This may be because of redistribution of the ratios of ventilation to perfusion throughout the lungs as may be expected considering the surgical manoeuvres performed which also change total pulmonary blood flow by altering cardiac output. These changes in ventilation and perfusion can produce changes in  $V_D^{phys}$  which are reflected by changes in  $P(a-E')CO_2$ .  $P_{aCO_2}$  cannot be predicted from  $P_{E'}CO_2$  in patients with changing lung conditions.<sup>16</sup>

In conclusion, patients with mitral stenosis have higher  $P(a-E')CO_2$  and lower  $P_{aO_2}$ , which may necessitate higher  $FI_{O_2}$ .  $P_{E'}CO_2$  does not accurately reflect  $P_{aCO_2}$  during the surgical procedure of close mitral commissurotomy. Similar findings are expected in any patient group with changing pulmonary characteristics under anaesthesia.

### References

1. TAKKI S, AROMAA U, KAUSTE A. The validity and usefulness of the end-tidal  $PCO_2$  during anaesthesia. *Annals of Clinical Research* 1972; 4: 278-84.
2. FLETCHER R. The single breath test for  $CO_2$ . Thesis, University of Lund, Sweden, 1980.
3. NUNN JF, HILL DW. Respiratory dead space and arterial to end-tidal  $CO_2$  tension difference in anaesthetized man. *Journal of Applied Physiology* 1960; 15: 383-9.
4. WHITESELL R, ASIDDAO C, GOLLMAN D, JABLONSKI J. Relationship between arterial and peak expired carbon dioxide pressure during anaesthesia and factors influencing the difference. *Anesthesia and Analgesia* 1981; 60: 508-12.
5. RAEMER DB, FRANCIS D, PHILIP JH, GABEL RA. Variation in  $PCO_2$  between arterial blood and peak expired gas during anaesthesia. *Anesthesia and Analgesia* 1983; 62: 1065-9.

6. GABEL RA. Algorithms for calculating and correcting blood-gas and acid-base variables. *Respiratory Physiology* 1980; **42**: 211–32.
7. FLETCHER R, WERNER O, NORDSTRÖM L, JOHNSON B. Sources of error and their correction in the measurement of carbon dioxide elimination using the Siemens-Elema CO<sub>2</sub> analyzer. *British Journal of Anaesthesia* 1983; **55**: 177–85.
8. PAVANKUMAR P, VENUGOPAL P, KAUL U, IYER KS, DAS B, SAMPATHKUMAR A, AIRON B, RAO IM, SHARMA ML, BHATIA ML, GOPINATH N. Closed mitral valvotomy during pregnancy. A twenty-year experience. *Scandinavian Journal of Thoracic and Cardiovascular Surgery* 1988; **22**: 11–15.
9. RAINE J, BISHOP JM. The distribution of alveolar ventilation in mitral stenosis at rest and after exercise. *Clinical Science* 1963; **24**: 63–8.
10. COLLINS JV, CLARK TJH, BROWN DJ. Airway function in healthy subjects and patients with left heart disease. *Clinical Science and Molecular Medicine* 1975; **49**: 217–28.
11. SUTHERLAND PW, CADE JF, PAIN MCF. Pulmonary extravascular fluid volume and hypoxaemia in myocardial infarction. *Australian and New Zealand Journal of Medicine* 1971; **1**: 141–5.
12. TATTERSFIELD AE, MCNICOL MW, SILLETT RW. Relationship between haemodynamic and respiratory function in patients with myocardial infarction and left ventricular failure. *Clinical Science* 1972; **42**: 751–68.
13. INTERIANO B, HYDE RW, HODGES M, YU PN. Interrelation between alterations in pulmonary mechanics and hemodynamics in acute myocardial infarction. *Journal of Clinical Investigation* 1973; **52**: 1994–2006.
14. DEMEDTS M, SNIDERMAN A, UTZ G, PALMER WH, BECKLAKE MR. Lung volumes including closing volume, and arterial blood gas measurements in acute ischaemic left heart failure. *Bulletin de Physio-pathologie Respiratoire* 1974; **10**: 11–25.
15. NUNN JF. *Applied respiratory physiology*, 2nd edn. London: Butterworth, 1977.
16. YAMANAKA MK, SUE DY. Comparison of arterial-end-tidal PCO<sub>2</sub> difference and dead space/tidal volume ratio in respiratory failure. *Chest* 1987; **92**: 832–5.

*Anaesthesia*, 1991, Volume 46, pages 496–499

### Antagonism of atracurium with neostigmine

#### Effect of dose on speed of recovery

D. R. Goldhill, FFARCS, Senior Lecturer, \*J. A. Carter, FFARCS, Senior Registrar, D. Suresh, FFARCS, J. P. Whitehead, FFARCS, Senior Registrars, P. J. Flynn, FFARCSI, Senior Lecturer, The Anaesthetics Unit, The London Hospital, London E1 1BB.

#### Summary

*In 36 patients in whom anaesthesia was maintained with nitrous oxide and 0.5% isoflurane an atracurium-induced neuromuscular block was either allowed to recover spontaneously or antagonised with one of four doses of neostigmine (15 µg/kg, 35 µg/kg, 55 µg/kg or 75 µg/kg). The recovery times to a train-of-four ratio of 0.5, 0.75 and 0.9 were recorded. In patients given neostigmine, antagonism was at an average T1 of between 8.8% and 14.9%. There was no difference in the recovery times between the patients given neostigmine 35 µg/kg, 55 µg/kg or 75 µg/kg. Recovery after neostigmine 15 µg/kg was significantly slower than after the higher doses. One patient given neostigmine 75 µg/kg showed an unusual bimodal pattern of recovery. There appears to be no benefit in giving a larger dose than 35 µg/kg of neostigmine as a single bolus.*

#### Key words

*Antagonists; neostigmine.  
Monitoring; train-of-four stimulation.  
Neuromuscular relaxants; atracurium.*

The anticholinesterase neostigmine is commonly administered at completion of surgery to antagonise residual paralysis from nondepolarising neuromuscular blocking agents. If too little antagonist is given, recovery of muscle power will be longer than necessary and such patients may have residual paralysis on admission to the recovery area.<sup>1–3</sup> Too large a dose of antagonist may, at least theoretically, increase muscle weakness.<sup>4</sup> The optimum dose will be the smallest dose that results in the speediest return of muscle power.

Spontaneous recovery of muscle power may be

prolonged after atracurium<sup>5</sup> and even a small dose of neostigmine (0.625 mg) will speed recovery.<sup>6</sup> Although a modest dose of neostigmine (1.25 mg) may sometimes be adequate,<sup>6</sup> studies examining a range of neostigmine dose found that the highest dose administered (50 µg/kg)<sup>7,8</sup> resulted in fastest recovery. There is little information on the effect of a dose of neostigmine greater than 50 µg/kg when used to antagonise an atracurium-induced neuromuscular block, but there is a plateau to its action after pancuronium; 80 µg/kg is no more effective than 60 µg/kg.<sup>9</sup>

The present study was designed to identify the optimum

\*Now Consultant, Chelmsford, Essex.

Accepted 26 August 1990.

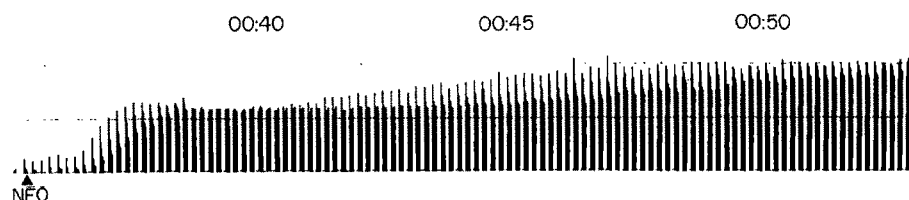


Fig. 1. Relaxograph trace showing bimodal pattern of recovery in a patient given neostigmine 75 µg/kg.

dose of neostigmine required to antagonise a profound atracurium-induced neuromuscular blockade.

### Methods

Ethics committee approval was obtained and written consent given by the 36 patients in the study. All were ASA 1 undergoing elective surgery. Patients taking medication known to interfere with neuromuscular function were not studied.

Premedication comprised intramuscular papaveretum (15–20 mg) with hyoscine (0.3–0.4 mg). Anaesthesia was induced with fentanyl (1–2 µg/kg) and thiopentone (4–6 mg/kg) and maintained with oxygen, nitrous oxide (66%) and 0.5% inspired isoflurane (measured with a Datex Normac). Ventilation was controlled to maintain an end-tidal  $P_{aCO_2}$  of 4.6 to 5.3 kPa. A Datex Relaxograph was used to provide supramaximal stimulation of the ulnar nerve via silver/silver-chloride surface electrodes at the wrist. Relaxograph placement was distributed at random between the arms. The impulses were of 0.1 ms duration and delivered as a train of four (TOF) at 2 Hz every 10 seconds. The Relaxograph was used to measure the integrated evoked compound electromyogram (EMG) using similar electrodes placed over the *adductor pollicis* muscle. A recording of the output for later analysis was made onto a Gould 220 chart recorder. The arm from which record-

ings were taken was wrapped in cotton wool and palm temperature was maintained at 34–37°C.

After induction of anaesthesia a stable neuromuscular response was established and a single bolus dose of atracurium (0.4 mg/kg or 0.35 mg/kg) was administered. The neuromuscular response was allowed to recover spontaneously until three consecutive TOF stimuli evoked two twitches in response (point R). Patients were then allocated at random to receive one of four doses of neostigmine in combination with glycopyrronium: group 1, neostigmine 15 µg/kg; group 2, 35 µg/kg; group 3, 55 µg/kg; and group 4, 75 µg/kg.

Four patients constituted a control group. They were studied using an identical protocol but muscle power was allowed to recover spontaneously. They have not been included in the statistical analysis. In all groups the anaesthesia was continued throughout the period of recovery.

Control twitch ( $T_c$ ) was defined as the  $T_1$  of the TOF when the TOF ratio was 0.9. Note was made of the  $T_1/T_c$  at antagonism, the  $T_1/T_c$  and TOF ratio every minute for 10 minutes after antagonism and the time to achieve a TOF ratio of 0.5, 0.75 and 0.9. Relevant times for the control group are from when three consecutive stimuli evoked a response of two twitches (point R).

Statistical analysis was performed using the BMDP statistical package. Significance of differences in recovery times to TOF ratios of 0.5, 0.75 and 0.9 were assessed using one-way analysis of variance (ANOVA) for all four treatment groups and for the three groups given the larger doses of neostigmine (35 µg/kg, 55 µg/kg and 75 µg/kg). Where a significant difference was found the Student–Newman–Keuls (S–N–K) test was used to identify differences between the groups. A  $p < 0.05$  was accepted as significant.

### Results

One of the patients who was given neostigmine 75 µg/kg exhibited a bimodal pattern of recovery in which initial recovery was followed by an increase in  $T_1$  followed by further recovery of  $T_4$  (Fig. 1). This patient has been excluded from statistical analysis. A further patient, who received 15 µg/kg of neostigmine, was only monitored until

Table 1. Patients' characteristics.

Group	n	Age; years (SEM)	Weight; kg (SEM)	Sex M/F
1; neostigmine 15 µg/kg	8	27.3 (3.2)	60.6 (1.8)	2/6
2; neostigmine 35 µg/kg	8	30.5 (3.5)	68.2 (3.6)	4/4
3; neostigmine 55 µg/kg	8	27.6 (3.6)	64.5 (2.7)	2/6
4; neostigmine 75 µg/kg	8	29.3 (2.6)	70.8 (4.3)	3/5
5; control	4	29.5 (4.0)	66.5 (3.5)	2/2

Table 2. Recovery data.

	Group (numbers in each group in parenthesis)				
	1 (8)	2 (8)	3 (8)	4 (7)	Control (4)
$T_1/T_c$ % at antagonism (SEM)	11.5 (1.94)	14.9 (2.63)	11.0 (1.02)	8.8 (0.8)	6.3* (0.9)
Time; seconds, to TOF ratio=0.5 (SEM)	585 (75.5)	289 (27.3)	244 (38.0)	259 (33.2)	1720 (268)
Time; seconds, to TOF ratio=0.75 (SEM)	809 (64.5)	451 (33.2)	395 (68.2)	434 (47.4)	2130 (231)
Time; seconds, to TOF ratio=0.9 (SEM)	987† (73.3)	618 (45.8)	608 (84.0)	599 (63.3)	2680 (203)

\*,  $T_1/T_c$  at point R, (three consecutive stimuli evoking two twitches).

†,  $n=7$ .

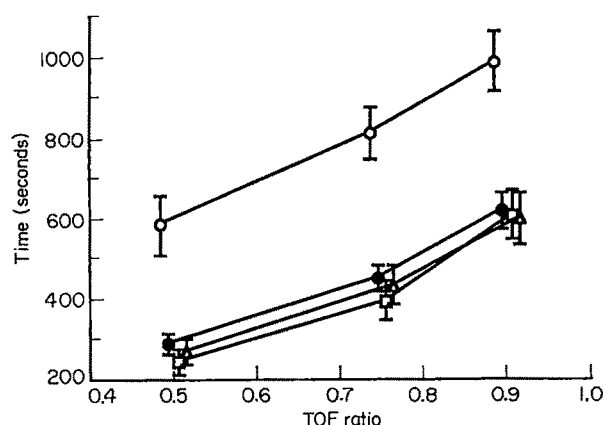


Fig. 2. Time (seconds) after neostigmine administration to reach TOF ratios of 0.5, 0.75 and 0.9 (○, 15 µg/kg, ●, 35 µg/kg, □, 55 µg/kg, or △, 75 µg/kg).

the TOF ratio was 0.87. The T1 at this point was taken as the Tc. There was no significant difference between the groups with regard to age, weight, sex distribution (Table 1), or the T1/Tc at antagonism (Table 2). Mean time (SEM) from the initial bolus of atracurium until point R for patients who received neostigmine was 36.6 (1.28) minutes for nine patients given atracurium 0.4 mg/kg, and 30.5 (1.24) minutes for the other patients who received 0.35 mg/kg.

An average (SEM) of 23 seconds (1.9) elapsed from point R until neostigmine was administered. Figure 2 shows the times taken after administration of neostigmine for twitch height to reach a TOF ratio of 0.5, 0.75 and 0.9. Figures 3 and 4 represent the data in another way showing the T1/Tc% (Fig. 3), and the TOF ratio (Fig. 4), every minute for the 10 minutes after reversal.

There was a significant difference in times to target TOF ratios between groups 1, 2, 3 and 4 ( $p = 0.0001$  for TOF 0.5,  $p < 0.0001$  for TOF 0.75,  $p = 0.001$  for TOF 0.9). The S-N-K test showed a significant difference between group 1 (neostigmine 15 µg/kg) and the other three groups. There was no significant difference by ANOVA between groups 2, 3 and 4 (neostigmine 35 µg/kg, 55 µg/kg and 75 µg/kg) in the time taken to a TOF ratio of 0.5 ( $p = 0.62$ ), 0.75 ( $p = 0.73$ ) and 0.9 ( $p = 0.98$ ). In the recovery area clinical recovery of muscle power, determined by head lift and hand grip, was satisfactory in all patients.

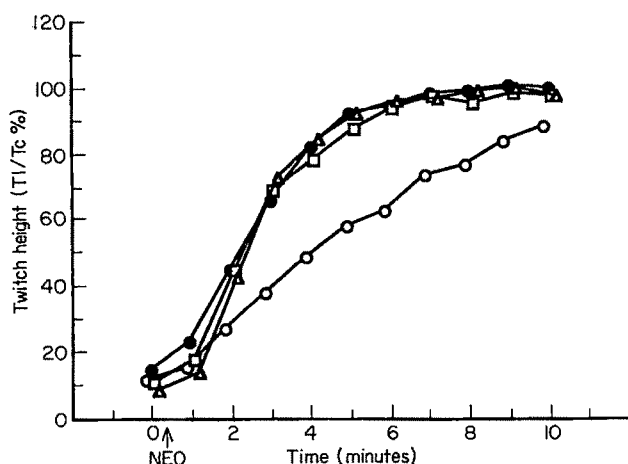


Fig. 3. T1/Tc% every minute for 10 minutes after administration of neostigmine (○, 15 µg/kg, ●, 35 µg/kg, □, 55 µg/kg, or △, 75 µg/kg). Neostigmine administered at NEO.

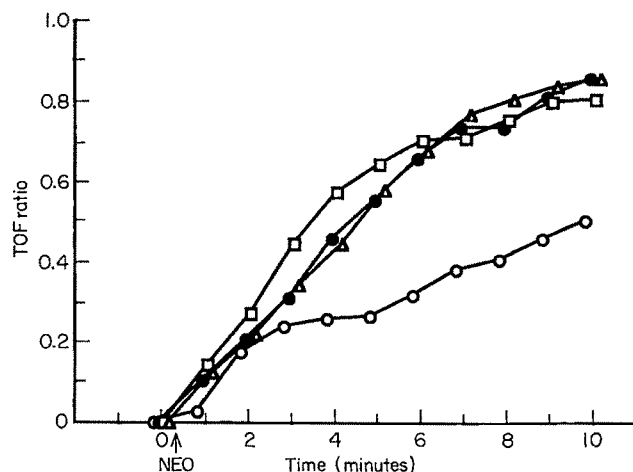


Fig. 4. TOF ratio every minute for 10 minutes after administration of neostigmine (○, 15 µg/kg, ●, 35 µg/kg, □, 55 µg/kg, or △, 75 µg/kg). Neostigmine administered at NEO.

### Discussion

In order to standardise the recovery characteristics during the study and in view of the possibility of baseline drift with the Relaxograph recording, we related all neuromuscular function to TOF characteristics. We therefore chose to antagonise at the point where two twitches of the TOF first appeared rather than rely on T1 height. To be certain that this point had been consistently achieved we waited until three consecutive stimuli produced this response before administering the neostigmine. The results show that there is no difference between the groups in the height of T1/Tc at this point. At a TOF of 0.9 the T1 has reached a plateau and is at its maximum. We therefore took the T1 at this time as our control twitch (Tc).

In the immediate postoperative period incomplete recovery of neuromuscular function is possible even with the medium duration relaxants, atracurium and vecuronium,<sup>3</sup> and is a potential cause of postoperative morbidity.<sup>10,11</sup> Adequacy of recovery of neuromuscular function is a clinical decision. However in uncooperative or unconscious patients a clinical assessment is not possible and a TOF ratio of approximately 0.75 has been shown to correlate with signs of adequate recovery.<sup>12</sup> The relevant TOF ratio for atracurium may be less and it has been suggested that a figure of 0.5 may be acceptable.<sup>13</sup> The TOF ratio may also vary depending on whether neuromuscular function is measured electromyographically or mechanically,<sup>14</sup> with the EMG recovering before the mechanical response. Recent work has shown that previously accepted limits may be inadequate<sup>15</sup> and conservative management may therefore dictate that a TOF ratio of 0.9 is reached to be certain that muscle power has recovered.<sup>14</sup> We therefore chose as our end points TOF ratios of 0.5, 0.75 and 0.9, each of which might be accepted as a measure of adequate recovery.

Too small a dose of neostigmine increases the risk of inadequate recovery of muscle power. Too large a dose appears to be no more effective than a smaller dose and may, at least theoretically, diminish muscle power.<sup>4</sup> We have defined the optimum dose of anticholinesterase as the smallest dose which will achieve the fastest recovery of neuromuscular function. Many factors may affect the optimum dose or the time taken for antagonism to occur. These include the relaxant used, the anaesthetic technique and the degree of spontaneous recovery at reversal. If the neuromuscular block is profound then recovery may be

prolonged no matter what dose of neostigmine is administered.<sup>16</sup> If recovery is nearly complete then little neostigmine may be required to overcome residual block. If no antagonist is administered, spontaneous recovery as shown by our control group, is prolonged. Even a small dose of neostigmine (15 µg/kg) will accelerate recovery, but the larger doses (35 µg/kg, 55 µg/kg and 75 µg/kg) are more effective.

Previous work concluded that neostigmine 50 µg/kg<sup>7,8</sup> provided faster recovery than 20 µg/kg. Our work shows that increasing the dose of neostigmine to more than 35 µg/kg does not speed reversal under the conditions of our study. Indeed, there is a plateau to the effectiveness of neostigmine and there appears to be no benefit from increasing the dose further. Neostigmine 75 µg/kg was no more effective than the smaller doses and one subject showed an unusual pattern of reversal (Fig. 1). In this patient there was a rapid initial recovery of fade followed by a return of T1 to pre-relaxant control height, once again followed by recovery of fade. The time taken after antagonism to finally reach a TOF ratio of 0.9 was longer than 16 minutes in this patient. The actions of neostigmine are complex with effects at pre and postsynaptic receptors as well as at other sites, and although neostigmine will usually antagonise a block it may also cause fade.<sup>4</sup> The trace seen in this patient may be caused by the differential action of neostigmine at pre and postsynaptic sites, or may show a direct detrimental effect on neuromuscular function.

In conclusion, neostigmine accelerates recovery from an atracurium-induced block when spontaneous recovery to a T1/Tc of about 12% has occurred. The speed of recovery increases until a dose of neostigmine 35 µg/kg is reached and then reaches a plateau with no apparent differences between 35 µg/kg, 55 µg/kg and 75 µg/kg. When antagonising such an atracurium-induced blockade at least 35 µg/kg of neostigmine should be administered, but there appears to be no benefit in giving a larger dose as a single bolus.

## References

1. VIBY-MOGENSEN J, JORGENSEN BC, ORDING H. Residual curarization in the recovery room. *Anesthesiology* 1979; **50**: 539–41.
2. BEEMER GH, ROZENTAL P. Postoperative neuromuscular function. *Anaesthesia and Intensive Care* 1986; **14**: 41–5.
3. BEVAN DR, SMITH CE, DONATI F. Postoperative neuromuscular blockade: a comparison between atracurium, vecuronium, and pancuronium. *Anesthesiology* 1988; **69**: 272–6.
4. GOLDBILL DR, WAINWRIGHT AP, STUART CS, FLYNN PJ. Neostigmine after spontaneous recovery from neuromuscular blockade. Effect on depth of blockade monitored with train-of-four and tetanic stimuli. *Anaesthesia* 1989; **44**: 293–9.
5. ERKOLA O, KARHUNEN U, SANDELIN-HELQVIST E. Spontaneous recovery of residual neuromuscular blockade after atracurium or vecuronium during isoflurane anaesthesia. *Acta Anaesthesiologica Scandinavica* 1989; **33**: 290–4.
6. JONES JE, PARKER CJR, HUNTER JM. Antagonism of blockade produced by atracurium or vecuronium with low doses of neostigmine. *British Journal of Anaesthesia* 1988; **61**: 560–4.
7. SMITH CE, DONATI F, BEVAN DR. Dose-response relationships for edrophonium and neostigmine as antagonists of atracurium and vecuronium neuromuscular blockade. *Anesthesiology* 1989; **71**: 37–43.
8. DONATI F, SMITH CE, BEVAN R. Dose-response relationships for edrophonium and neostigmine as antagonists of moderate and profound atracurium blockade. *Anesthesia and Analgesia* 1989; **68**: 13–19.
9. GOLDBILL DR, EMBREE PB, ALI HH, SAVARESE JJ. Reversal of pancuronium. Neuromuscular and cardiovascular effects of a mixture of neostigmine and glycopyrronium. *Anaesthesia* 1988; **43**: 443–6.
10. LUNN JN, HUNTER AR, SCOTT DB. Anaesthesia-related surgical mortality. *Anaesthesia* 1983; **38**: 1090–6.
11. COOPER AL, LEIGH JM, TRING IC. Admissions to the intensive care unit after complications of anaesthetic techniques over 10 years. 1. The first 5 years. *Anaesthesia* 1989; **44**: 953–8.
12. ALI HH, KITZ RJ. Evaluation of recovery from non-depolarizing neuromuscular block, using a digital neuromuscular transmission analyzer: preliminary report. *Anesthesia and Analgesia Current Researches* 1973; **52**: 740–4.
13. JONES RM, PEARCE AC, WILLIAMS JP. Recovery characteristics following antagonism of atracurium with neostigmine or edrophonium. *British Journal of Anaesthesia* 1984; **56**: 453–7.
14. KOPMAN AF. The relationship of evoked electromyographic and mechanical responses following atracurium in humans. *Anesthesiology* 1985; **63**: 208–11.
15. PAVLIN EG, HOLLE RH, SCHOENE RB. Recovery of airway protection compared with ventilation in humans after paralysis with curare. *Anesthesiology* 1989; **70**: 381–5.
16. CALDWELL JE, ROBERTSON EN, BAIRD WLM. Antagonism of profound neuromuscular blockade induced by vecuronium or atracurium. *British Journal of Anaesthesia* 1986; **58**: 1285–9.



### Serum bupivacaine concentrations following wound infiltration in children undergoing inguinal herniotomy

K.A. Mobley, FFARCS, J.G. Wandless, FFARCS, Consultants, D. Fell, FFARCS, Senior Lecturer, Department of Anaesthesia, Leicester Royal Infirmary, Leicester LE1 5WW, A. McBurney, PhD, Department of Chemical Pathology, Glenfield General Hospital, Leicester LE3 9QP.

#### Summary

*Serum bupivacaine concentrations were measured in 12 children who underwent elective herniotomy and who received analgesia in the form of wound infiltration. Mean (SD) peak concentration was 0.36 (0.14) µg/ml and time to peak concentration was 14.6 (7.2) minutes after infiltration of 1.25 mg/kg of bupivacaine. These concentrations are lower than those associated with other local anaesthetic blocks and well below potentially toxic levels. Wound infiltration provides a simple, effective and safe method of providing postoperative analgesia for hernia repair in children.*

#### Key words

*Anaesthetics, local; bupivacaine.*

*Anaesthetic technique; wound infiltration.*

Local anaesthetic techniques are used widely to provide postoperative analgesia in children who undergo day-case herniotomy. Wound infiltration with bupivacaine has been shown to provide analgesia comparable to that associated with caudal block<sup>1</sup> or ilioinguinal block.<sup>2</sup> Bupivacaine may produce toxic effects at plasma concentrations greater than 4 µg/ml<sup>3,4</sup> although the rate of increase of plasma concentration is an important factor.<sup>5,6</sup> Both caudal and ilioinguinal blocks have been shown to result in concentrations well below 4 µg/ml.<sup>7</sup> Bupivacaine concentrations have been measured following subcutaneous injection,<sup>8</sup> but this does not mimic the conditions of wound infiltration and this information is not available for this age group.

The purpose of this study was to measure serum bupivacaine concentrations after wound infiltration for postoperative analgesia in children who underwent day-case herniotomy.

#### Method

The study was approved by the District ethics committee and informed consent was obtained from one or both parents. Twelve children aged between 2 and 10 years admitted for elective unilateral herniotomy as day-cases were studied. No premedication was given and general anaesthesia was induced by inhalation of halothane or by the intravenous route (following application of EMLA cream). Maintenance of anaesthesia was by spontaneous ventilation of 67% nitrous oxide in oxygen and halothane delivered by a T-piece or Bain coaxial system.

A 20-gauge cannula was inserted into a vein on the dorsum of the hand either for intravenous induction or after inhalational induction, and was used for blood sampling. A baseline (2 ml) sample was taken prior to surgery. Before skin closure, the wound edges were infiltrated with 1.25 mg/kg of bupivacaine without adrenaline as either the 0.25% solution in children under 16 kg or as the 0.5% solution in those of greater weight. Further venous blood samples were taken at 5, 10, 15, 20, 30, 45

and 60 minutes after the completion of wound infiltration. The patency of the cannula was maintained with heparinised saline and the deadspace was aspirated on each occasion. Samples were placed in plain tubes and centrifuged to yield approximately 1 ml of serum which was stored at –70°C.

Bupivacaine concentrations were measured by high pressure liquid chromatography using dichloromethane for the extraction and p-chlorodisopyramide as internal standard. Interassay coefficient of variation was 5.1% and intra-assay coefficient of variation was less than 10%.

#### Results

The ages and weights of patients are shown in Table 1. A complete set of results was not obtained for all patients because of sampling difficulties. The serum bupivacaine concentrations in one patient were somewhat higher than the others. Consequently, the median and range of bupivacaine concentrations are presented in Table 2. The mean (SD) maximum concentration of 0.36 (0.14) µg/ml was reached at 14.6 (7.2) minutes.

#### Discussion

The results of this study demonstrate that venous serum bupivacaine concentrations below 0.7 µg/ml are found in children who receive herniotomy wound infiltration with 1.25 mg/kg of bupivacaine. Tucker and colleagues<sup>9</sup> showed

Table 1. Mean (SD) and range of ages and weights of patients.

Age (years)	Weight (kg)
5.1 (2.1)	18.0 (3.8)
2.2–8.5	13.5–25.0

Table 2. Median and range of serum bupivacaine concentrations ( $\mu\text{g/ml}$ ) after wound infiltration.

Minutes	0	5	10	15	20	30	45	60
Median	0	0.21	0.27	0.27	0.34	0.33	0.28	0.17
Range	0	0.14–0.69	0.17–0.65	0.21–0.55	0.21–0.52	0.17–0.58	0.10–0.55	0.14–0.34
n	12	12	11	12	11	10	10	9

binding to be the same in plasma and serum and expected no difference in drug concentration between plasma and serum. Our results are therefore comparable with other studies in which plasma concentrations have been measured.

Arterial concentrations may be up to 30% higher than venous concentrations<sup>10,11</sup> but allowing for this, the highest concentration in our group of patients would be less than  $1 \mu\text{g/ml}$  and well below the widely quoted figure of  $4 \mu\text{g/ml}$ <sup>3,4</sup> at which toxic manifestations may appear.

The time to peak concentration was 14.6 minutes (95% confidence intervals 9.8–19.4 minutes). This did not appear to be related to concentration of solution used although patient numbers were inadequate for statistical analysis. It is our usual practice to use bupivacaine 0.25% in a dose which is often less than  $0.5 \text{ ml/kg}$ , depending on the size of the incision. However, for the purposes of the study we used a standard weight-related dose which necessitated the use of 0.5% solution in the larger children. Concentrations were higher in one patient than in the others, but the peak concentration was only  $0.69 \mu\text{g/ml}$ , attained after 5 minutes.

Local anaesthetic techniques are used commonly for hernia repair in children, particularly for day-case surgery, to avoid the use of systemic opioids. A previous study from this department<sup>1</sup> has shown that wound infiltration is as effective as caudal block in providing analgesia for inguinal herniotomy in children. Serum bupivacaine concentrations reached with wound infiltration are similar or below those measured by other authors after caudal block<sup>7,8,10,12</sup> or ilioinguinal block.<sup>7</sup> Bupivacaine dosage varies between studies and recommended maximum dosage has little meaning without reference to site of injection.<sup>13</sup> The most likely cause of a toxic reaction is intravascular injection. A significant intravascular injection is least likely with wound infiltration, as the end of the needle is constantly moving during the injection.

Despite the small numbers in this study we believe that we have demonstrated the safety of this simple method of postoperative analgesia in children in respect of serum bupivacaine concentrations.

## References

1. FELL D, DERRINGTON MC, TAYLOR E, WANDLESS JG. Paediatric postoperative analgesia. A comparison between caudal block and wound infiltration of local anaesthetic. *Anaesthesia* 1988; **43**: 107–10.
2. REID MF, HARRIS R, PHILLIPS PD, BARKER I, PEREIRA NH, BENNETT NR. Day-case herniotomy in children. A comparison of ilio-inguinal nerve block and wound infiltration for postoperative analgesia. *Anaesthesia* 1987; **42**: 658–61.
3. JORFELDT L, LÖFSTRÖM B, PERNOW B, PERSSON B, WAHREN J, WIDMAN B. The effect of local anaesthetics on the central circulation and respiration in man and dog. *Acta Anaesthesiologica Scandinavica* 1968; **12**: 153–69.
4. MOORE DC, BALFOUR RI, FITZGIBBONS D. Convulsive arterial plasma levels of bupivacaine and the response to diazepam therapy. *Anesthesiology* 1979; **50**: 454–6.
5. SCOTT DB. The evaluation of the toxicity of local anaesthetic agents in man. *British Journal of Anaesthesia* 1975; **47**: 56–61.
6. SCOTT DB. Evaluation of clinical tolerance of local anaesthetic agents. *British Journal of Anaesthesia* 1975; **47** Suppl.: 328–331.
7. STOW PJ, SCOTT A, PHILLIPS A, WHITE JB. Plasma bupivacaine concentrations during caudal analgesia and ilioinguinal-iliohypogastric nerve block in children. *Anaesthesia* 1988; **43**: 650–3.
8. EYRES RL, KIDD J, OPPENHEIM R, BROWN TCK. Local anaesthetic plasma levels in children. *Anaesthesia and Intensive Care* 1978; **6**: 243–7.
9. TUCKER GT, BOYES RN, BRIDENBAUGH PO, MOORE DC. Binding of anilide-type local anaesthetics in human plasma. *Anesthesiology* 1970; **33**: 287–314.
10. EYRES RL, BISHOP W, OPPENHEIM RC, BROWN TCK. Plasma bupivacaine concentrations in children during caudal epidural analgesia. *Anaesthesia and Intensive Care* 1983; **11**: 20–2.
11. MOORE DC, MATHER LE, BRIDENBAUGH PO, BRIDENBAUGH LD, BALFOUR RI, LYSONS DF, HORTON WG. Arterial and venous plasma levels of bupivacaine following epidural and intercostal nerve blocks. *Anesthesiology* 1976; **45**: 39–45.
12. ECOFFEY C, DESPARMET J, MAURY M, BERDEAUX A, GIUDICELLI J-F, SAINT-MAURICE C. Bupivacaine in children: pharmacokinetics following caudal anaesthesia. *Anesthesiology* 1985; **63**: 447–8.
13. SCOTT DB. 'Maximum recommended doses' of local anaesthetic drugs. *British Journal of Anaesthesia* 1989; **63**: 373–4.

## Correspondence

<b>Pain after surgery</b>	502	<b>Failure of hydrocortisone to prevent postoperative sore throat</b>	512
<i>J.A. Jones, FRCP, FFARCS and A.W. Harrop-Griffiths, FFARCS</i>		<i>D.V. Thomas, MD, FFARCS</i>	
<i>A.A. Spence, FCAnaes</i>	504	<i>P.C. Stride, FFARCS</i>	512
<b>Impairment of ventricular diastolic function during coronary artery bypass grafting</b>	504	<b>Hypoxaemia during induction of anaesthesia</b>	513
<i>K. Muralidhar, MD</i>		<i>A. Gupta, FFARCS, M. Vegfors, MD and C. Lennmarken, MD, PhD</i>	
<b>Regional anaesthesia and cough effectiveness</b>	504	<b>Factors that influence the induction dose of propofol</b>	513
<i>D.A. Conn, FCAnaes, A.C. Moffat, FFARCSI and R.A. Duckworth, FCAnaes</i>		<i>G.A. McLauchlan, MRCP, FCAnaes, P. Spiers, FCAnaes and J.E. Peacock, FCAnaes</i>	
<i>A.W. Harrop-Griffiths, FFARCS, A. Ravalia, FFARCSI, D.A. Browne, FFARCSI and P.N. Robinson, FFARCS</i>	505	<i>G.J. McClean, FFARCS</i>	513
<b>Malfunction of a Laerdal resuscitation valve</b>	505	<b>Hyoscine derivative in children—but was it hyoscine?</b>	514
<i>W.A.H. Hunter, FCAnaes and R.A. Duthie</i>		<i>J. Rupprecht, MD</i>	
<i>K.G. Morallee</i>	506	<b>Emergency adenotonsillectomy for acute airway obstruction</b>	514
<b>Pressure generated during insertion of lumbar epidurals</b>	506	<i>G. Francis, FFARCS</i>	
<i>S.R. Hayes, FFARCSI</i>		<i>N. Solomons, FRCS</i>	515
<i>C.G. Peters</i>	506	<i>R.J. Harwood, FCAnaes, T.A. King, FCAnaes, D.F. Johnston, FRCS and J.C. Watkinson, FRCS</i>	515
<i>J.A. Langton, FFARCS, and B.H. Meiklejohn, FFARCS</i>	507	<b>Does complicated mean better?</b>	515
<b>The serotonin syndrome</b>	507	<i>A. Gilston, FFARCS</i>	
<i>T.K. Ooi, FCAnaes</i>		<b>Faulty Portex epidural needle</b>	516
<b>An isolated grand mal seizure 5 days after propofol anaesthesia</b>	508	<i>D.A. Cozanites, MD</i>	
<i>J.S. Thomas, FFARCS and N.O. Boheimer</i>		<i>N. Green</i>	516
<b>An interesting misconnection</b>	508	<b>A retrospective analysis of epidural vein trauma</b>	516
<i>C. Hayes, FCAnaes</i>		<i>A.L. Skidmore, FFARCS</i>	
<b>Bronchospasm following application of bilateral lower limb tourniquets</b>	509	<b>Three in one block for fractured neck of femur</b>	517
<i>M.S. Sammut, MRCP</i>		<i>A.G. Jones, MB, BS</i>	
<b>Bimanual cricoid pressure—another version</b>	510	<b>Dilatation of hand veins</b>	517
<i>R. Williamson, FFARCS</i>		<i>D.V.A. Kalmanovitch, FFARCS</i>	
<b>Routine pre-oxygenation</b>	510	<b>Pulse oximetry in the postcardiopulmonary bypass period</b>	517
<i>G. Skea, MB, ChB, A. Jones, MB, BS and S.L. Snowdon, FFARCS</i>		<i>D.E. Withington, MRCP, FFARCS</i>	
<b>Fluid balance in ARDS</b>	511	<b>Anaesthesia for cataract surgery</b>	518
<i>I. McConachie, FFARCS</i>		<i>J.E. Goold, FCAnaes</i>	
<b>Acupuncture and postoperative sickness</b>	512	<b>An alternative throat spray</b>	518
<i>J.W. Dundee, MD, PhD, FFARCS</i>		<i>M.R. Nott, FFARCS</i>	
		<b>Withdrawal of levorphanol</b>	518
		<i>S.W. Coniam, FCAnaes</i>	
		<b>Dr Gordon Knowles</b>	519
		<i>A.K. Mathur, FFARCS</i>	

### Pain after surgery

We are sure that many of your readers will have read with interest the recent report of a Working Party of the Colleges of Surgeons and Anaesthetists on Pain after Surgery.<sup>1</sup> The summary of this publication begins 'The management of pain after surgery in the U.K. is unsatisfactory'. On page 5 it is stated that 'many studies have shown that a significant number of patients experience an

unacceptable degree of pain after surgery when it is treated with conventional intramuscular opioid therapy'. These statements conflict with our own clinical experience and understanding of the literature, so we took the opportunity to read the 14 papers which were quoted in support of these statements. What follows is a brief comment on each of these papers. The figure in brackets is that quoted by the

All correspondence should be addressed to Dr M. Morgan, Editor of *Anaesthesia*, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, United Kingdom.

Letters must be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters contained in the Notice to Contributors to *Anaesthesia* printed at the back of each issue. The degrees and diplomas of each author must be given in a covering letter personally signed by all the authors.

Correspondence presented in any other style or format may be the subject of considerable delay and may be returned to the author for revision. *If the letter comments on a published article in Anaesthesia, please send three copies; otherwise two copies of your letter will suffice.*

report as being the proportion of patients with 'insufficient analgesia' or in 'moderate or severe pain' indentified in each paper.

Papper *et al*, 1954<sup>2</sup> (33%). In this study the severity of postoperative pain was inferred from the frequency with which the patients received postoperative opioid injections. As there was no attempt to assess the efficacy of their pain treatment this data cannot be used to support the premise that intramuscular opioids are often ineffective.

Lasagna and Beecher, 1954<sup>3</sup> (33%). This paper reported a trial in which subcutaneous injections of morphine were administered. The pharmacokinetics of drugs given by this route differ from those of intramuscular injections.

Keats, 1965<sup>4</sup> (33%). This study also employed subcutaneous morphine.

Keeri-Szanto & Heaman, 1972<sup>5</sup> (20%). In this trial patients were given morphine, pentazocine or oxymorphone by an unspecified route. The use of the latter two drugs cannot be considered to be 'conventional', and as the paper did not state what proportion of the patients received each drug, the use of these data in this context must be treated with some suspicion.

Cronin *et al*, 1973<sup>6</sup> (42%). This paper claimed that 42% of patients who had undergone upper abdominal surgery and who received intramuscular morphine or pethidine postoperatively experienced pain that was 'very unpleasant indeed' or worse. The patients were not premedicated and did not receive intra-operative analgesia or indeed any inhalational anaesthetic agent other than nitrous oxide. It is not surprising therefore that a significant proportion of patients who underwent partial gastrectomy, cholecystectomy, hiatus hernia repair, laparotomy or adrenalectomy felt pain.

Banister, 1974<sup>7</sup> (12–26%). Twenty-six percent of patients complained of pain after piritramide, 21% after levorphanol and 15% after pentazocine; these are not 'conventional' opioids. One hour after pethidine or papaveretum the proportion of patients reporting moderate or severe pain was 14%, i.e. 86% reported no pain or slight pain.

Tammisto, 1978<sup>8</sup> (24%). This trial studied subjective analgesia after intramuscular oxycodone. The authors noted that 'even after laparotomies, less than 20% (of patients) had severe complaints concerning the efficacy of pain relief'.

Cohen, 1980<sup>9</sup> (75%). This paper reported that 75% of patients who had undergone abdominal surgery and who had received intramuscular pethidine (in unspecified amounts) had a 'moderate or marked pain distress score'. This score assessed eight different complaints only one of which was adequacy of pain relief. The other complaints included depression, irritability and anger. The authors noted that '79.8% of patients said that (pain) relief was adequate'.

Tamsen *et al*, 1982<sup>10</sup> (38%). In a trial that was neither blind, controlled nor randomised, 58 patients received opioids via a PCA device postoperatively. The figure quoted in the report comes from the following sentence: 'Postoperative intramuscular analgesia given at the patient's request after termination of PCA was considered very effective by 62% and incomplete by 38%. The authors did not report which analgesics were used, the doses or how many of the 58 patients received them.

Donovan B, 1983<sup>11</sup> (31%). In this study 200 patients undergoing surgery were given either intramuscular pethidine or papaveretum; 86% of patients were satisfied with their postoperative pain relief. The figure of 31% is acquired by adding 21 patients who were not satisfied with their pain relief to 41 patients who were satisfied but admitted to feeling some pain.

Weis *et al*, 1983<sup>12</sup> (41%). This paper studied 66 patients

who received opioids after 'elective major surgery'. We are not told which drug was given or in what doses. To quote the authors: 'Only 18% thought postoperative pain relief had been inadequate'.

Donovan M, 1987<sup>13</sup> (58%). Fifty-eight percent of 454 medical and surgical patients reported that they had experienced moderate or severe pain during their admission. The paper does not relate how many of these patients had undergone surgery or what analgesic drugs were given by which route and in what doses. The authors commented that 'All patients reporting severe pain indicated that pain medications were beneficial in reducing the pain'.

Seers, 1989<sup>14</sup> (43%). Eighty percent of patients who had undergone abdominal surgery were visited twice a day for their first postoperative week and were invited to assess their pain on a 'verbal rating scale' which ranged from 'no pain' through 'slight pain', 'quite a lot of pain' and 'very bad pain' to 'agonising pain'; 43% of patients had 'quite a lot of pain' or worse in the first postoperative day; 65% of patients admitted that analgesics improved their pain and 61% considered that pain control had been as good as or better than they had expected. The techniques of pain relief employed are not described.

Owen *et al*, 1990<sup>15</sup> (37%). Of the 57% of 213 patients who had received intramuscular morphine or pethidine, 37% reported that they had felt 'severe or unbearable pain' within the first 24 hours of surgery; 79% of patients said that the effectiveness of their 'painkillers' had been moderate or better. It is worthy of note that at a pre-operative visit 'more than two-thirds (of the patients) said they would wait until they were in severe pain before requesting analgesia'. The authors commented that 'analgesics were generally effective'.

In reporting the above observations we do not seek to ridicule the report of the Working Party or to pour scorn on its recommendations, many of which are cogent and relevant. We do believe, however, that the above suggests that there are insufficient data relating to the efficacy of currently employed postoperative analgesia regimens to justify the widespread adoption of analgesic techniques which, although of a potentially greater efficacy, may involve a substantially greater risk to the patient. The report's comment that 'research into pain relief after surgery should be encouraged and intensified' is to be wholly supported, however, this research should include not only that into new techniques, but also research into the adequacy and safety of currently employed techniques so that new developments can be compared with data that have been accurately reported from well-conducted trials.

St. Mary's Hospital,  
London W2 1NY

J.A. JONES  
A.W. HARROP-GRIFFITHS

## References

1. Report of the Working Party on Pain After Surgery Commission on the Provision of Surgical Services. The Royal College of Surgeons of England/The College of Anaesthetists, 1990.
2. PAPPER, EM, BRODIE BB, ROVENSTINE EA. Postoperative pain: its use in the comparative evaluation of analgesics. *Surgery* 1954; **32**: 303–7.
3. LASAGNA L, BEECHER HK. The optimal dose of morphine. *Journal of the American Medical Association* 1954; **15**: 230–4.
4. KEATS AS. Postoperative pain: research and treatment. *Journal of Chronic Diseases* 1956: 472–83.
5. KEERI-SZANTO M, HEAMAN S. Postoperative demand analgesia. *Surgery, Gynecology and Obstetrics* 1972; **134**: 647–51.
6. CRONIN M, REDFERN PA, UTTING JE. Psychometry and postoperative complaints in surgical patients. *British Journal of Anaesthesia* 1973; **4**: 879–86.

7. BANISTER EHD. Six potent analgesic drugs. A double-blind study in postoperative pain. *Anaesthesia* 1974; **29**: 158–62.
8. TAMMISTO T. Analgesics in postoperative pain relief. *Acta Anaesthesiologica Scandinavica* 1978; (Suppl. 70) **22**: 47–50.
9. COHEN FL. Postsurgical pain relief: patients' status and nurses' medication choices. *Pain* 1980; **9**: 265–74.
10. TAMSEN, A. Comparison of patient-controlled analgesic therapy: clinical experience. *Acta Anaesthesiologica Scandinavica* (Suppl. 74) 1985; **26**: 157–60.
11. DONOVAN BD. Patient attitudes to postoperative pain relief. *Anaesthesia and Intensive Care* 1983; **11**: 125–9.
12. WEIS OF SRIWANTANAKUL K, ALLOZA JL, WEINTRAUB M, LASAGNA L. Attitudes of patients, housestaff and nurses toward postoperative analgesic care. *Anesthesia and Analgesia* 1983; **62**: 70–4.
13. DONOVAN M, DILLON P, MCGUIRE, L. Incidence and characteristics of pain in a sample of medical-surgical inpatients. *Pain* 1987; **30**: 69–78.
14. SEERS, K. Patients' perception of acute pain. In: WILSON-BARNETT J, ROBINSON S, eds. *Directions in nursing research*. London: Scutari Press, 1989; 107–16.
15. OWEN H, MCMILLAN, V, ROGOWSKI, D. Postoperative pain therapy: a survey of patients' expectations and their experiences. *Pain* 1990; **41**: 303–7.

#### A reply

I cannot accept the broad criticisms of your correspondents, but they are correct in drawing attention to

the fact that subcutaneous, not intramuscular, injection was used in some of studies cited. I apologise for this error which was the result of over-enthusiastic editing. The main point is that unacceptable degrees of pain occur in the post-operative period. This is supported by the studies we listed and has been re-asserted recently.<sup>1</sup> It is also worth reporting that a number of lay people have written to commend the Colleges' initiative with recollection of their own unfavourable experience.

Practice varies between hospitals and thus the good results that Drs Jones and Harrop-Griffiths allude to are reassuring. I would urge them to follow the injunction to greater audit of practice because I think there are many who would benefit from their lead. We would stand by our criticism of 'traditional' methods but that is not to say that the drugs are useless or that the route and rate of administration cannot be improved.

Chairman, COPSS Working Party  
on Pain after Surgery,  
College of Anaesthetists,  
Royal College of Surgeons of England,  
London WC2A 3PN

A.A. SPENCE

#### Reference

1. KUHN S, COOKE K, COLLINS M, JONES JM, MUCKLOW JC. Perceptions of pain relief after surgery. *British Medical Journal* 1990; **300**: 1687–90.

#### Impairment of ventricular diastolic function during coronary artery bypass grafting

I was interested to read the above paper (*Anaesthesia* 1990; **45**: 549–51), but believe that some important relevant information has not been mentioned. What was the nature of pre-operative drug therapy? The question whether the impaired diastolic function after bypass could be related to residual effects of the pre-operative drugs, such as  $\beta$ -adrenoceptor blocking agents and calcium antagonists, has to be answered. Did all the patients receive similar pre-operative drug therapy? Eight patients in the study had pre-operative hypertension; was the phenomenon of impairment of diastolic function more marked in the hypertensive patients? I would expect a greater degree of diastolic dysfunction in hypertensives as a result of chronic hypertrophy of the left ventricle.

Can the good diastolic function before surgical incision be attributed to the effect of anaesthesia alone? The decreased systemic oxygen requirement seen under anaesthesia provides a general protective effect for patients with limited cardiac reserve.<sup>1</sup> Benzodiazepines produce a 'nitroglycerine' like effect on coronary vasculature, which preserves coronary blood flow.<sup>2</sup> Etomidate, which has been used in the study, is a drug that changes the haemodynamic variables the least and hence produces least change in the balance of myocardial oxygen demand supply.<sup>3</sup> I believe that the combination of agents used for induction in this study offered many therapeutic benefits, namely decreased

sympathetic activity, haemodynamic stability, and enhanced coronary flow.

Is it possible that the diastolic dysfunction was present pre-operatively, was ameliorated by anaesthesia, and then was unmasked at the end of surgery? Did the authors use any inotropes postbypass? Can the impairment of diastolic dysfunction at the end of surgery indicate subclinical coronary vasospasm? What was the method of myocardial preservation and duration of aortic cross clamping?

I believe these questions require answering before any conclusions can be drawn with regard to coronary artery bypass grafting and impairment of diastolic function.

B.M. Birla Heart Research Centre, K. MURALIDHAR  
Calcutta 700 027,  
India

#### References

1. MERRIN RG. Effects of anesthesia on heart. *Surgical Clinics of North America* 1975; **55**: 759–74.
2. COTE P, GUERET P, BOURASSA MG. Systemic and coronary hemodynamic effects of diazepam on patients with normal and diseased coronary arteries. *Circulation* 1974; **50**: 1210–16.
3. REVES JG, FLEZZANI P, KISSIM I. Pharmacology of intravenous anesthetic. Induction drugs. In: Kaplan JA, ed. *Cardiac anesthesia*. Orlando: Grune and Stratton, 1987: 136.

#### Regional anaesthesia and cough effectiveness

We were interested to read the report by Harrop-Griffiths *et al.* (*Anaesthesia* 1991; **46**: 11–13). They described the effects of spinal and epidural anaesthesia on respiratory variables during Caesarean section.

The measurements were only taken before the start of surgery. As they point out in the discussion, it is likely that the abdominal muscles are the most important muscles in the production of an effective cough. Would it not

therefore have been a more clinically relevant comparison to assess the pre-operative values with those taken once the abdomen was opened? In pregnancy respiratory function is already compromised by assumption of the supine position compared to the erect. We therefore fail to see any relevance in measuring preblock erect values and comparing them to supine postblock results. In our opinion they have failed to show any clear or convincing evidence based on the figures they present to support their hypothesis that a patient's ability to cough may be significantly impaired if there is an inadvertently high block. Finally to make a link with a case of acid aspiration during epidural anaesthesia for Caesarean section in a patient with pre-existing pulmonary disease would appear tenuous.

Western Infirmary,  
Glasgow G11 6NT

D.A. CONN  
A.C. MOFFAT  
R.A. DUCKWORTH

### Reply

We are grateful to Drs Conn, Moffat and Duckworth for their interest in our paper, and to the Editor for giving us the opportunity to reply. We address their comments in turn. Ethical considerations restricted our measurements to the pre-incision period; however, we would agree that a further reduction in the parameters measured may have been seen had we performed measurements once the abdomen had been opened. Gamil<sup>1</sup> recorded the lowest values of peak expiratory flow in his trial on patients undergoing Caesarean section under regional anaesthesia when the patient had an open abdomen.

We have been able to confirm the common assumption that respiratory function is compromised in the parturient when lying supine. Our comparison with preblock erect values is relevant not only because it is in this position that respiratory function tests are usually performed, but also because it allowed us to compare the relative effects of change in position and regional anaesthesia on the parameters measured.

We believe that we have shown that a parturient's capacity to cough effectively may be impaired by regional anaesthesia. It is reasonable to assert that this is largely a result of motor blockade, and that the more extensive the motor blockade the greater the extent to which cough effectiveness may be impaired. It is therefore not unreasonable to suggest that an inadvertently high block may bring this impairment into the realm of clinical significance.

We were careful to say that the occurrence of acid aspiration during regional anaesthesia for Caesarean section in a patient with pre-existing pulmonary disease 'may lend credence to' our hypothesis. Drs Conn, Moffat and Duckworth may consider this link to be tenuous, but this does not mean that it may not have some clinical relevance.

St. Mary's Hospital,  
London W2 1NY  
Edgware General Hospital,  
Edgware, Middlesex

A.W. HARROP-GRIFFITHS  
A. RAVALLIA  
D.A. BROWNE  
P.N. ROBINSON

### Reference

1. GAMIL M. Serial peak expiratory flow rates in mothers during Caesarean section under extradural anaesthesia. *British Journal of Anaesthesia* 1989; 62: 415-8.

### Malfunction of a Laerdal resuscitation valve

We report an incident arising from the malfunction of a Laerdal resuscitation valve.

A 52-year-old man was admitted to the Intensive Therapy Unit following triple coronary artery bypass grafting for elective ventilation. His condition was stable and at 0500 hours he was disconnected from the ventilator to allow adjustment of the ventilator tubing. A Laerdal resuscitation bag and valve which had previously been used without problems was connected to the tracheal tube. Inflation of the lungs rapidly became more difficult and the ITU resident anaesthetist was summoned. On arrival he found the patient in a state of imminent cardiac arrest. His heart rate was 25 beats/minute and arterial blood pressure 40/20 mmHg. His chest appeared hyperinflated and he had surgical emphysema on the left side of his neck. On removal of the Laerdal bag and valve the lungs deflated and tracheal suction confirmed tube patency. Atropine 1 mg was administered intravenously and external cardiac massage commenced. This was followed by adrenaline 1 mg intravenously and the sternotomy wound was re-opened to permit internal cardiac massage. Ventilation was continued with 100% oxygen with a different Laerdal bag and valve and was easy with good air entry bilaterally. Within 5 minutes of the initial episode the patient had responded to the resuscitation. His arterial blood pressure was 180/90 mmHg, heart rate 120 beats/minute and central venous pressure 6 mmHg. He had an uneventful course following resuscitation and was discharged from ITU 12 hours afterwards. He left hospital a week later with no obvious sequelae.

After the incident the Laerdal bag and valve was

examined. On initial inspection the assembly appeared normal (Fig. 1), but in use was found to act as a one-way valve, preventing expiration. Closer examination revealed that the valve contained not one but two rubber diaphragms which were so closely adherent that they appeared as a single unit (Fig. 2). When either of the diaphragms was removed the valve functioned normally. This problem must have arisen from the practice of cleaning and re-using the diaphragms which were stored stacked together before re-insertion. New diaphragms are packed individually but are still regarded as re-usable, a practice which could lead to repetition of the problem. We checked all the Laerdal bags in the hospital following this incident and found no further problems.

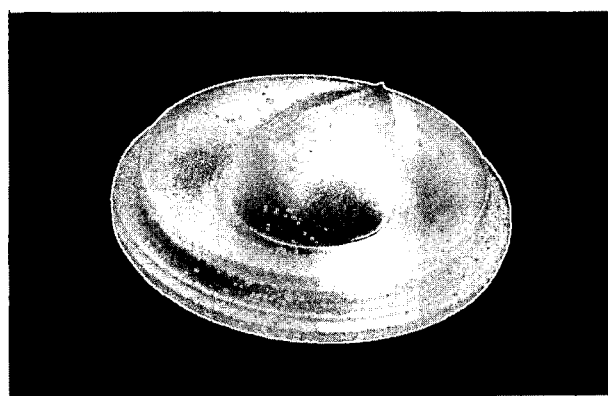


Fig. 1. The apparently normal diaphragm.

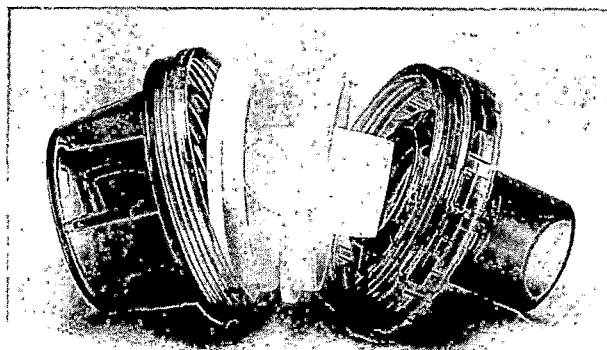


Fig. 2. The assembly on further inspection.

Laerdal International and the Scottish Home and Health Department have been informed and a hazard warning should be issued in the near future.

Royal Infirmary,  
Aberdeen AB9 2ZB

W.A.H. HUNTER  
R.A. DUTHIE

#### A reply

Thank you for the opportunity of replying to the letter from Drs Hunter and Duthie.

In order for the Laerdal resuscitator to function properly the lip membrane valve must lift during expiration. The presence of the second valve prevented this in the incident reported by Dr Hunter. This fault should have been detected by function testing after reassembly as described in our wall poster and the 'Directions for Use' supplied with each resuscitator. The Department of Health has advised on a number of occasions the importance of staff training: instructions must be known and followed by those who take apart and reassemble emergency equipment, and we are pleased to provide free of charge posters and additional copies of our 'Directions for Use' for this purpose.

However, we are examining modifications to the valve to obviate the possibility of such misassembly occurring.

Managing Director,  
Laerdal Medical Ltd  
Orpington, Kent BR6 0HX

K.G. MORALLEE

#### Pressure generated during insertion of lumbar epidurals

I read with interest the paper by Drs Langton and Meiklejohn (*Anaesthesia* 1990; **45**: 1055-6.) This study confirms that the Portex epidural injection simulator requires generation of similar mean pressures within the 'detector syringe,' to those generated *in vivo*. The discrepancy between the pressures generated when air or saline are used reflects a difference in compressibility. The authors do not state the size of the syringe used in their study. This is of relevance when interpreting their results, since the pressure generated within a syringe by an identical force is inversely proportional to the diameter of the syringe barrel. To reduce the risk of inadvertent dural puncture it is important to minimise any force transmitted through the tip of the Tuohy needle. It would have been interesting to have calculated the forces applied to the syringe plunger as well as the pressures generated.

Ideally, when locating the epidural space using a loss-of-resistance technique, a constant force should be applied to the detector device, i.e. the syringe plunger. In one of the authors' references, pressure tracings illustrate that widely fluctuant pressures were generated during epidural space location.<sup>1</sup> Was this the case in this study? It would have been of interest to know the peak pressures obtained during epidural insertion. The authors do not describe the technique of epidural space identification used, but the method described by Doughty<sup>2</sup> is alluded to<sup>3</sup> in their discussion. Was this technique used during this study?

In their discussion the authors mention that considerable force may be necessary to advance a Tuohy needle through the ligamentum flavum, thus implying (erroneously) that the magnitude of this force is related to that applied to a syringe plunger when the loss-of-resistance technique is used. The texture and resistance of the ligamentum flavum to the passage of a Tuohy needle may vary greatly between individuals, on occasion very little force being necessary. It is therefore important to optimise sensitivity in detection of loss of resistance. In achieving this goal, use of the palm of the hand and the palmar surface of the metacarpophalangeal joint at the base of the index finger, to apply a constant force to the syringe plunger, as described by Doughty, is beneficial; this is the area which is sensitive to

pressure change on the hand, as is manifest in location of an elusive cardiac apex beat.

Victoria Infirmary,  
Glasgow G42 0TY

S.R. HAYES

#### References

1. SHARROCK NE. Recordings of, and an anatomical explanation for, false positive resistance during lumbar extradural analgesia. *British Journal of Anaesthesia* 1979; **51**: 253-8.
2. DOUGHTY A. LETTER. *Anaesthesia* 1982; **37**: 470.
3. MACDONALD R. Dr Doughty's technique for the location of the epidural space. *Anaesthesia* 1983; **38**: 71-2.

I was interested in the observation by Drs Langton and Meiklejohn (*Anaesthesia* 1990; **45**: 1055-6) that the pressure generated in locating the extradural space was lower with air than with saline. No explanation was offered for this.

I wonder if in locating the extradural space the six anaesthetists used a continuous advancing and testing technique, or an intermittent one. I, perhaps like many, use a technique of continuous pressure on the syringe plunger and advance the needle at the same time, rather than intermittently advancing then testing. Writing as a convert from air to saline, I find that a noncompressable fluid gives a more distinct 'loss of resistance' than air. The pressure generated in locating the extradural space is produced by thumb pressure on the plunger of the syringe. I use a combination of this pressure on the plunger of the syringe together with pressure on the needle hub in order to advance the needle through ligamentum flavum. However, I would tend to favour an intermittent technique when testing with air because of a less distinct 'loss of resistance'. Might this account for the difference in generated pressure between air and saline?

The authors suggest that the reported lower dural puncture rate when locating the extradural space with saline may be due to a lower incidence of needle blockage.



Could it also be that the loss of pressure of about 1000 mmHg is more easily appreciated to an 'educated' thumb than the loss of pressure of 115 mmHg?

New Cross Hospital,  
Wolverhampton WV10 0QP

C.G. PETERS

#### A reply

Thank you for the opportunity of replying to the letters from Dr S.R. Haynes and from Dr C.G. Peters.

It is important to be clear which pressures are being studied. The pressure measured in our study is that generated within the medium (air or saline) contained within the syringe. It is this pressure which constitutes the resistance which is 'lost' on entering the epidural space. This pressure varies greatly depending on whether air or saline is used and reflects the differing compressibility of the medium.

This pressure is also dependant upon the size of the syringe barrel being inversely proportional *not* to the diameter but to the square of the radius.<sup>1</sup> This variable was eliminated from our study by using the Portex *loss of resistance* device as supplied in the Portex Minipack kits for all the measurements. These forces and pressures are quite

different from those involved in the actual advancement of the needle through the tissues. Dr Haynes is correct to state that to minimise this force would reduce the risk of inadvertent dural puncture, but this force was not the subject of our study, although its investigation would be interesting, but technically challenging.

The loss of resistance technique used with air was an intermittent 'test-advance' method. The pressure generated fluctuated from zero during advancement to 210 mmHg during testing. The method using saline was essentially that described by Doughty,<sup>2</sup> whereby the syringe and needle are advanced with simultaneous continuous pressure on the plunger.

We agree with Dr Peters that the loss of 1000 mmHg pressure when using saline should be more easily appreciated than the more subtle changes occurring when air is used. We also believe that a higher chance of needle blockage using air may contribute to the dural puncture rate.

Leicester Royal Infirmary,  
Leicester LE1 5WW

J.A. LANGTON  
B.H. MEIKLEJOHN

#### References

1. PARBROOK GD, DAVIS PD, PARBROOK EO. Basic physics and measurement in anaesthesia. London: Heinemann 1982: 2-3.
2. MACDONALD R. Dr Doughty's technique for the location of the epidural space. *Anaesthesia* 1983; 38: 71-2.

#### The serotonin syndrome

The serotonin syndrome is due to the accumulation of excess 5 Hydroxytryptamine (5HT or serotonin) in the brain. This condition has been extensively studied in animals.<sup>1-3</sup>

A 72-year-old woman with chronic depression was admitted following acute onset of confusion. She had been receiving fluoxetine 60 mg/day but this was changed to Parstelin (a combination of tranylcypromine 10 mg and trifluoperazine 1 mg) twice daily. On day two of her new medication she became feverish, was shivering, rigid, developed a tremor and was admitted because of deteriorating conscious level. On examination, she was delirious, her axillary temperature was 40°C and she had a tachycardia. She opened her eyes spontaneously and withdrew to painful stimuli. There was marked rigidity in all four limbs. Reflexes and plantar responses were difficult to assess. There were no other physical signs.

The British National Formulary (BNF) was consulted as fluoxetine was a new drug. A boxed warning in the book described serotonin syndrome as a side-effect from interaction between fluoxetine and monoamine oxidase inhibitors (MAOIs).<sup>4</sup> The other differential diagnoses were neuroleptic malignant syndrome (NMS) and septicæmia. The features of NMS are fever, extrapyramidal rigidity, disturbances of autonomic function and unconsciousness. It occurs more often with high potency and depot neuroleptic drugs and 3-9 days after initiation of therapy or a change in dosage of neuroleptics. NMS is commonly complicated by rhabdomyolysis resulting in renal failure, intravascular coagulation with pulmonary embolism and respiratory failure.<sup>5</sup> None of these were present in the patient described. NMS was unlikely since she became symptomatic after two doses of trifluoperazine and there was no autonomic instability. Blood and urinary cultures were taken to exclude septicæmia. After routine baseline investigations, the only abnormality was a white cell count of  $18 \times 10^9/\text{litre}$ .

She was sedated with intravenous diazepam 20 mg and paralysed with pancuronium, which was chosen because it would prevent the hypotensive effect of serotonin syndrome. Her lungs were artificially ventilated via an oro-tracheal tube. Phenytoin 1 g was given to prevent arrhythmias and she was sedated with an infusion of midazolam. Cooling was achieved with cold intravenous fluids and icepacks and after 2 hours her temperature had decreased to 38°C. Six hours later her temperature was normal. It remained normal for 2 days and on the third day she was weaned from artificial ventilation and her trachea was extubated. Over the next 6 hours, she again became confused, rigid and her temperature rose to 39°C. She was resedated with diazepam and re-paralysed with pancuronium and her trachea was re-intubated. After a further 2 days, weaning from artificial ventilation proved to be no problem. Urinary culture grew group  $\beta$  haemolytic streptococci, sensitive to cefotaxime. Blood cultures proved negative.

Other treatments include withdrawal of possible offending drugs and symptomatic treatment of other abnormalities. Additional treatments described were methysergide (5HT antagonist)<sup>2,6</sup> and propranolol<sup>7</sup>, which were not used since she responded immediately to IPPV.

It was unlikely that septicæmia played a part as blood cultures were negative and this patient's pyrexia was corrected with muscle paralysis and IPPV.

Fluoxetine was launched as a new antidepressant in the UK in 1989.<sup>8</sup> It selectively inhibits the reuptake of 5HT by postsynaptic nerve terminals resulting in an accumulation in the brain.<sup>9</sup> Because of the long half-lives of fluoxetine and norfluoxetine (its active metabolite), MAOIs should not be prescribed until fluoxetine has been discontinued for at least 5 weeks. Similarly, MAOIs should be discontinued for at least 2 weeks before starting treatment with fluoxetine. Deaths in patients started on MAOI shortly after ceasing fluoxetine have been reported.<sup>4,8</sup>

With MAOIs back in vogue, clinicians should be aware of their more unusual interactions with other drugs. As anaesthetists we may be called to resuscitate patients with the serotonin syndrome; muscle paralysis and IPPV are effective treatment in most patients.

St. George's Hospital,  
London SW17 0QT

T.K. Ooi

### References

- JACOBS BL. An animal behavior model for studying central serotonergic synapses. *Life Sciences* 1976; **19**: 777-86.
- SQUIRES RF. MAOI: Animal pharmacology. In: IVERSEN L, IVERSEN S, SNYDER S, eds. *Handbook of psychopharmacology*, Vol 14.
- GERSON SC, BALDESSARINI RJ. Motor effects of serotonin in the central nervous system. *Life Sciences* 1980; **27**: 1435-51.
- KLINE SS, MAURO LS, SCALA-BARNETT DM, ZICK D. Serotonin syndrome versus neuroleptic malignant syndrome as a cause of death. *Clinical Pharmacy* 1989; **8**: 510-4.
- PEARLMAN CA. Neuroleptic malignant syndrome: a review of the literature. *Journal of Clinical Psychopharmacology* 1986; **6**: 257-73.
- SANDYK R. L-dopa induced 'Serotonin Syndrome' in a parkinsonian patient on bromocriptine. *Journal of Clinical Psychopharmacology* 1986; **6**: 194-5.
- GUZE BH, BAXTER LR. The serotonin syndrome: case responsive to propranolol. *Journal of Clinical Psychopharmacology* 1986; **6**: 119-20.
- Drug and Therapeutics Bulletin* 1990; **28**: 33-4.
- SCHMIDT MJ, FULLER RW, WONG DT. Fluoxetine, a highly selective serotonin reuptake inhibitor: A review of preclinical studies. *British Journal of Psychiatry* 1988; **153** (Suppl. 3): 40-6.

### An isolated grand mal seizure 5 days after propofol anaesthesia

In the light of Saunders and Harris's interesting paper (*Anaesthesia* 1990; **45**: 552-7) we wish to report a case of a 27-year-old female who presented for termination of pregnancy as a day case. At pre-operative assessment, she appeared fit, did not volunteer any past medical history, took no medication and weighed 54 kg. Unpremedicated, the patient was anaesthetised with fentanyl 25 µg and propofol 100 mg and maintained with increments of propofol 10 mg whilst breathing spontaneously a mixture of nitrous oxide and oxygen. The patient was discharged from hospital 6 hours after a short uneventful procedure. Five days later, while at home, she developed myoclonic jerks that lasted for 10 hours, culminating in a single grand mal fit. Subsequently she revealed that she had suffered a generalised convulsion at the age of 12 years, and was treated for myoclonic attacks with sodium valproate until 1985, although she had been symptom free for the past 9 years. At the time of correspondence she has suffered no further fits or other neurological sequelae and has needed no treatment. As a consequence of the seizure, the patient's driving licence has been revoked, causing personal inconvenience and putting her job, as a travelling sales representative, in jeopardy.

The occasional unpredictable neuro-excitatory effects of propofol are well known. Up to May 1989, the Committee on Safety of Medicines (CSM) have received 37 reports of seizures of which 13 occurred in epileptics at induction or during early recovery from propofol anaesthesia; they recommend that care should be taken when administering the drug to epileptics.<sup>1</sup> It is difficult to establish a causal relationship between propofol and this isolated delayed fit, but it is equally hard to disprove it. The delay between drug administration and possible complication is particularly

worrying as the patient had been discharged from medical care. The mechanism by which propofol may have induced delayed fitting is unclear, but raises the possibility of an alternative or slower metabolic pathway in some patients. Saunders and Harris reported four female patients who suffered neurological sequelae after propofol anaesthesia, one of whom developed rapid jerky eyelid movements with no response to verbal command or pain, 34 hours after the procedure. The CSM have received eight reports of delayed recovery or relapsing into unconsciousness after the drug.<sup>1</sup>

Although propofol has been used in the management of status epilepticus,<sup>2</sup> this case highlights the danger of routine administration of this drug to patients with a history of epilepsy. We believe that propofol is contraindicated in epileptics and those with a previous history of the disorder, as a relapse of the condition can have deleterious social, economic and psychological consequences. Epilepsy is a disease with associated stigma, so patients may not readily volunteer information, as in this case. In the light of our experiences it seems prudent that anaesthetists specifically exclude a history of epilepsy before using propofol.

Singleton Hospital,  
Swansea SA2 8QA  
University Hospital of Wales  
Cardiff CF4 4XW

J.S. THOMAS  
N.O. BOHEIMER

### References

- Committee on Safety of Medicines. *Current problems* No. 26 May 1989.
- WOOD PR, BROWNE GPR, PUGH S. Propofol infusion for the treatment of status epilepticus. *Lancet* 1988; **1**: 480-1.

### An interesting misconnexion

A ratio of three anaesthetists to one patient contributed to this interesting misconnexion in a 54-year-old woman undergoing a mastectomy. Anaesthesia was induced in the anaesthetic room with alfentanil and thiopentone. Neuromuscular blockade was achieved using vecuronium and her trachea was intubated with an 8.0 mm orotracheal tube. Her lungs were ventilated without difficulty using a mixture of 30% oxygen in nitrous oxide and 1% enflurane. She was transferred to the operating theatre and ventilation continued with a Manley Pulmovent ventilator. Ventilation

pressure increased rapidly to 60 cmH<sub>2</sub>O. Manual ventilation was started without difficulty. There was good air entry bilaterally and no wheeze on auscultation of her chest. A problem with the ventilator was suspected. It was noted that the transfer tubing of the gas scavenge system was connected as in Fig. 1. The tubing ran from the Wright respirometer at the ventilator expiratory port to the expiratory valve of a Bain system. Connexion of the transfer tubing to the appropriate scavenge system inlet corrected the problem. Connexion to the expiratory valve of the Bain

SUCH A SENSITIVE AREA ...

"Headaches caused  
by leakage of  
spinal fluid may  
be minimised by  
using... a needle  
without a cutting  
bevel." <sup>1</sup>

NOW AVAILABLE  
IN 25G AND 27G

B-D® FINE GAUGE PENCIL POINT NEEDLES



... DESERVES A TRULY SENSITIVE APPROACH

**BECTON  
DICKINSON**

**Becton Dickinson UK Limited**

Between Towns Road, Cowley, Oxford, OX4 3LY, England.  
Tel: 0865 748844 Fax: 0865 717313

*Reference*

1. Moore DC. Regional Block. A handbook for use in clinical practice of medicine and surgery. 4th ed. Springfield III: Charles C Thomas; 1964; 358-359.

## THERE IS NO BETTER WAY TO STAY INFORMED

**Top authorities systematically review all advances**

## Experts select and evaluate key papers

**Comprehensive bibliography  
lists all papers  
in each area**

## Current Opinion in ANAESTHESIOLOGY

Reviews of all advances · Evaluation of key references  
Comprehensive listing of papers

Vol 4 1991 · No 1

No 1	THIS ISSUE Cardiovascular physiology and disease · P Barash Cardiac anaesthesia · NP de Bruijn Cardiovascular pharmacology · C Prys-Roberts	February
No 2	Intensive care · M Lamy    Resuscitation · N Birch	April
No 3	Obstetric anaesthesia and analgesia · TA Thomas Paediatric anaesthesia · J Lerman Anaesthesia and medical disease · BR Brown	June
No 4	Drugs in anaesthesia · M Wood Anaesthetics · D Lundberg    Opioids · PS Sebel Muscle relaxants · WC Bowman	August
No 5	Neurosurgical anaesthesia and neurological disease · W Fitch Regional anaesthesia · F Bonnet Pain therapy · P Raj	October
No 6	Practical anaesthesia · RF Moran Monitoring and equipment · A d'Hollander Pulmonary aspects of anaesthesia · JF Nunn	December

CS  
CURRICULUM  
SUMMARY

## C Prys-Roberts J G Reves

**NO RISK GUARANTEE:** You may cancel your subscription any time within 30 days of receiving your first issue and receive a full refund

## ORDER FORM

### Current Opinion in ANAESTHESIOLOGY

**Please enter my subscription to Volume 4, 1991 (6 issues):**

- ☐ **Personal** - from personal funds ONLY £75/\$145
- ☐ **Institutional** £150/\$255

**Subscription rates include airspeed delivery**  
**Subscriptions run on a calendar year basis**

- ☐ **30% discount for Students/ Housemen/ Residents**  
Deduct 30% from the prices above and provide proof of status

**Please send me further information on:**

- ☐ Current Opinion in ANAESTHESIOLOGY DATABASE
- ☐ The Annual of Cardiac Surgery
- ☐ Current Opinion in Radiology

**USA & Canada subscriptions to:** Current Science, 20 North 3rd Street, Philadelphia, PA 19106-2113, USA (USA & Canada subscriptions ONLY)  
**Rest of the world subscriptions to:** Current Science Ltd, Subscriptions, 34-42 Cleveland Street, London, W1P 5FB, UK

#### METHOD OF PAYMENT

- ☐ Am Ex   ☐ VISA   ☐ MasterCard   Amount payable . . . . .  
Card no |\_| |\_| |\_| |\_| |\_| |\_| |\_| |\_| |\_| |\_| |\_| |\_| Exp Date |\_| |\_| |\_|

Signature ..... Date .....

- ☐ Cheque/Eurocheque enclosed payable to Current Science Ltd  
☐ Bank transfer (Details available on request)

Payment may be made in US\$ at the current exchange rate  
 £ sterling must be drawn on a UK bank, US\$ on a US bank

- ☐ Please send an invoice (institutional subscriptions ONLY)

Name \_\_\_\_\_  
(BLOCK CAPITALS PLEASE)

**Address**

[illegible]

Return this form together with payment to address below

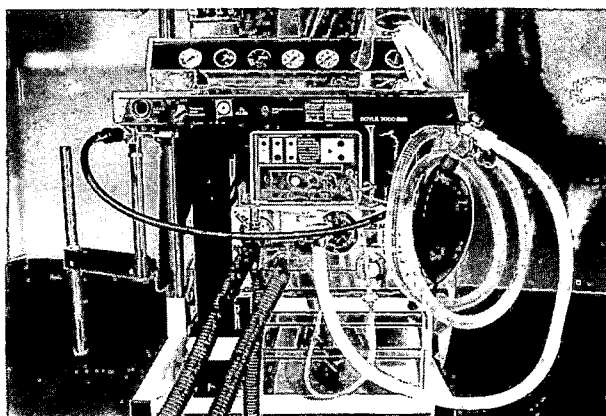


Fig. 1.

system had produced complete obstruction of the gas outflow from the ventilator.

The previous patient had been breathing spontaneously via a Bain system and it is suspected that the scavange system had been set up as in Fig. 2 with the transfer tubing running from the Bain system expiratory valve to the Wright respirometer. No problems had apparently been noted as expired gases passed out through the expiratory valve of the ventilator. It is thought that after completion of that case, the Bain system was disconnected from the fresh gas outflow of the anaesthetic machine but left with

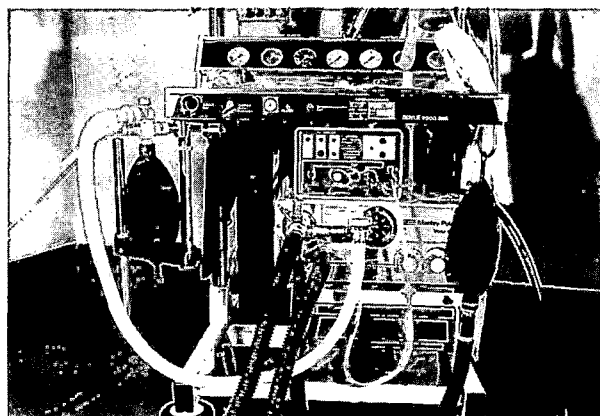


Fig. 2.

scavange transfer tubing attached. This produced the arrangement shown in Fig. 1.

The lessons illustrated by this episode are firstly to beware of multiple anaesthetists and secondly to check the gas scavange system in addition to other checks of anaesthetic machine function. None of the three anaesthetists present had checked the Manley Pulvovent circuit before the patient was transferred to the operating theatre.

Newcastle General Hospital,  
Newcastle upon Tyne NE4 6BE

C. HAYES

#### Bronchospasm following application of bilateral lower limb tourniquets

A number of adverse effects have been described in relation to the use of lower limb pneumatic tourniquets. The amount of blood and tissue fluid that is effectively squeezed out of both lower limbs using Esmarch bandages can range from 500 to 2000 ml and this is reflected in an immediate and sustained rise in central venous pressure of about 10 to 15 cm H<sub>2</sub>O.<sup>1,2</sup> Arterial hypertension can be observed in up to 11% of patients undergoing operations in which lower limb tourniquets are applied.<sup>3</sup> Dangerous rises in intracranial pressure in head injured patients have also been reported, both on tourniquet inflation and on deflation when reperfusion of the ischaemic limb results in an increased PaCO<sub>2</sub>.<sup>4,5</sup>

A fit, 43-year-old female non-smoker, weighing 52 kg, with a past history of allergic rhinitis, presenting for bilateral knee arthroscopies. At the patient's own request no premedication was prescribed. Anaesthesia was induced with 100 µg fentanyl and 150 mg propofol intravenously and a size 3 laryngeal mask was inserted. The patient was allowed to breathe spontaneously a mixture of nitrous oxide (4 litres/minute) and oxygen (2 litres/minute) and 2% enflurane via a Mapleson 'A' system. Both lower limbs were elevated and exsanguinated using Esmarch bandages and bilateral pneumatic tourniquets were applied. The patient's respirations became laboured, with a prolonged expiratory phase, and the neck veins distended. There was an audible expiratory wheeze at gas hose level and auscultation of the chest revealed widespread moderately severe bronchospasm. There were no inspiratory crepitations or gallop rhythm. The laryngeal mask was removed and ventilation with 100% oxygen and 3% enflurane was assisted via a facemask. The lungs were noted to be rather stiff. Terbutaline 0.5 mg was administered intravenously but very little improvement was seen. The tourniquets were both deflated and the bronchospasm very soon resolved completely and it was decided not to abandon the procedure. Both lower limbs were again exsanguinated and the tourniquets re-inflated. However, shortly before the first

arthroscopy was over, the patient again went into bronchospasm which did not respond to a further dose of 0.25 mg terbutaline but showed slight improvement when the first tourniquet was deflated. Complete resolution followed immediately on deflation of the second tourniquet. There were no postoperative sequelae.

Bronchoconstriction can be mediated via neural mechanisms predominantly involving myelinated vagal afferents, known as irritant receptors, which respond to both mechanical and chemical stimuli. Juxta capillary receptors or J-receptors, respond to pulmonary congestion, as can occur in left ventricular failure or fluid overload. In this patient bilateral lower limb exsanguination must have resulted in a substantial autotransfusion with a rise in central venous pressure seen clinically as neck vein distension. A subsequent rise in pulmonary capillary pressure may have caused activation of the J-receptors resulting in overt bronchospasm. This was largely unresponsive to the β-agonist given but responded immediately to tourniquet deflation, only to reappear on re-application. The presence of bronchospasm in acute pulmonary oedema is a well documented phenomenon and is thought to be due to stimulation of irritant receptors. Although there was no evidence at any stage that this patient was in pulmonary oedema it can be argued that a state of subclinical left ventricular failure was in fact present and that bronchospasm was the only clear manifestation.

69 Cheswick Drive,  
Gosforth,  
Newcastle upon Tyne NE3 5DW

M.S. SAMMUT

#### References

1. BOLISTON TA. Estimation of the exsanguinated volume of the leg by a gravimetric method. Proceedings of the Anaesthetic Research Society. *British Journal of Anaesthesia* 1986; 58: 123P.
2. BRADFORD EMW. Haemodynamic changes associated with the

- application of lower limb tourniquets. *Anaesthesia* 1969; 24: 190-7.
3. KAUFMAN RD, WALTS LF. Tourniquet-induced hypertension. *British Journal of Anaesthesia* 1982; 54: 333-6.
  4. ELDRIDGE PR, WILLIAMS S. Effect of limb tourniquet on

cerebral perfusion pressure in a head-injured patient. *Anaesthesia* 1989; 44: 973-4.

5. CONATY MC, KLEMM MS. Severe increase of intracranial pressure after deflation of a pneumatic tourniquet. *Anesthesiology* 1989; 71: 294-5.

#### Bimanual cricoid pressure—another version

The recent letter (*Anaesthesia* 1990; 45: 588-9) concerning bimanual cricoid pressure is a little confusing. Sellick described backward pressure on the cricoid cartilage which obliterated the oesophageal lumen at the level of the body of the fifth cervical vertebra in the supine patient whose 'head and neck are fully extended (as in the position for tonsillectomy)'. He made no mention of the sniffing position. On the other hand, Magill<sup>1</sup> had said that hyperextension, as used by some surgeons for bronchoscopy, actually increased the difficulty of intubation besides being unnecessary in most cases. He described the head slightly extended on the atlas and the cervical vertebrae not extended but in normal relation to the dorsal vertebrae, which might necessitate insertion of a pillow below the occiput as, '... in fact, are the relative positions of the air passages instinctively adopted by a man standing in the normal erect position when he scents the air.'<sup>1</sup>

Most intubations are probably performed in this 'Magill position', when the cricoid pressure will be applied at the level of the sixth cervical vertebra,<sup>2</sup> but workers investigating cricoid pressure<sup>3</sup> (except possibly Fanning)<sup>4</sup> have followed Sellick's example and employed the extended position. When Drs Crowley and Giesecke quote the 10.8N-120.6N range of cricoid pressures applied by assistants<sup>5</sup> and the force of 44N applied to the cricoid for effective protection in the majority of adults,<sup>3</sup> is it because they believe the methods and results exactly equate the real clinical situation? The former were derived from a test rig of a 60 ml syringe and a manometer, containing known volumes of air, connected by suitable tubing containing some water. For the latter,<sup>3</sup> patients had an 11 mm tube through the pharynx and a tracheal tube of undefined size through the larynx, and accompanying the clear trend of increasing intraluminal cricopharyngeal pressure (CPP) with increasing applied cricoid force (ACF) was a very wide scatter of CPP at each known ACF. The CPP when ACF was zero was higher than would be predicted from the other results. The tracheal tube exerting pressure, in the extended head-neck position,<sup>6</sup> could have been responsible thereby making all measurements unreliable clinically. Wraight *et al.*<sup>3</sup> suggested that 'statistically in their very small group', 44N offered 50% of patients protection against passive regurgitation and at 66N some 83% could expect protection. Certainly cricoid pressure is a useful means of increasing the safety margin during emergency induction of anaesthesia but regurgitation and aspiration

are of infrequent and irregular occurrence.<sup>7</sup> Without 100% effectiveness, or proof thereof, how is one certain of protecting all, or protecting those actually in need or, even, whom one is protecting? When the method, however ingenious, and the actual clinical situation differ the results should surely be considered useful but relative, rather than absolute, particularly if the significance of the differences is unknown.

Besides intravenous induction and tracheal intubation cricoid pressure is, perhaps, our most frequent high risk action where dangerous consequences can be closely linked to shortcomings of technique. More important than the question of one- or two-handed technique is that, in practice, it can never be taken for granted. The anaesthetist should give the technique the equal care and attention he would apply to his/her self. My preference is to stand on the right of the patient and apply cricoid pressure with my right hand. With my left hand on the patient's head to maintain the 'Magill position' I further assist the intubator by hooking up the right corner of the patient's mouth with my left thumb.

King Edward VIII Hospital,  
Durban  
South Africa

R. WILLIAMSON

#### References

1. MAGILL IW. Technique in endotracheal anaesthesia. *The British Medical Journal* 1930; ii: 817-20.
2. CHEEK TG, GUTCHE BB. Pulmonary aspiration of gastric contents. In: SHNIDER SM, LEVINSON G, eds. *Anesthesia for obstetrics*, 2nd edn. Baltimore. Williams & Wilkins 1987; 300-15.
3. WRAIGHT WJ, CHAMNEY AR, HOWELLS TH. The determination of an effective cricoid pressure. *Anaesthesia* 1983; 38: 461-6.
4. FANNING GL. The efficacy of cricoid pressure in preventing regurgitation of gastric contents. *Anesthesiology* 1970; 32: 553-5.
5. HOWELLS TH, CHAMNEY AR, WRAIGHT WJ, SIMONS RS. The application of cricoid pressure. *Anaesthesia* 1983; 38: 457-60.
6. LATTO IP. The cuff. In: LATTO IP, ROSEN M, eds. *Difficulties in tracheal intubation*. London: Baillière Tindall, 1985: 48-74.
7. OLSSON GL, HALLEN B, HAMBRAEUS-JONZON K. Aspiration during anaesthesia: a computer-aided study of 185,358 anaesthetics. *Acta Anaesthesiologica Scandinavica* 1986; 30: 84-92.

#### Routine pre-oxygenation

We would like to support the observations and conclusions concerning arterial oxygen saturation during the induction of anaesthesia recently reported by Drs Thorpe and Guantlett (*Anaesthesia* 1990; 45: 1012-5).

During a recent project to compare anaesthetic induction techniques for the purposes of medical audit, a surprisingly high incidence of oxygen desaturation was noted, (mean lowest figure of 86%) in a group of ASA 1 and 2 patients without any history of respiratory problems and whose

anaesthesia was administered by experienced registrars. It was therefore decided to assess the efficacy of pre-oxygenation before induction of mask anaesthesia. Forty-two ASA 1 or 2 patients were randomly allocated to two groups of 21. One group received a 'standard' anaesthetic defined as the most appropriate for that particular patient, whilst the second group received a similar anaesthetic but their lungs were pre-oxygenated. The latter was achieved by means of a Magill breathing



SUCH A SENSITIVE AREA ...

"Headaches caused  
by leakage of  
spinal fluid may  
be minimised by  
using a smaller  
gauge spinal  
needle ..." <sup>1</sup>

NOW AVAILABLE  
IN 27 AND 29  
GAUGE

B-D® FINE GAUGE SPINAL NEEDLES



... DESERVES PRECISION AT YOUR FINGER TIPS

**BECTON  
DICKINSON**

**Becton Dickinson UK Limited**

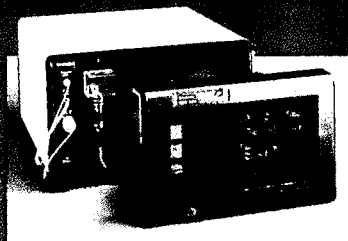
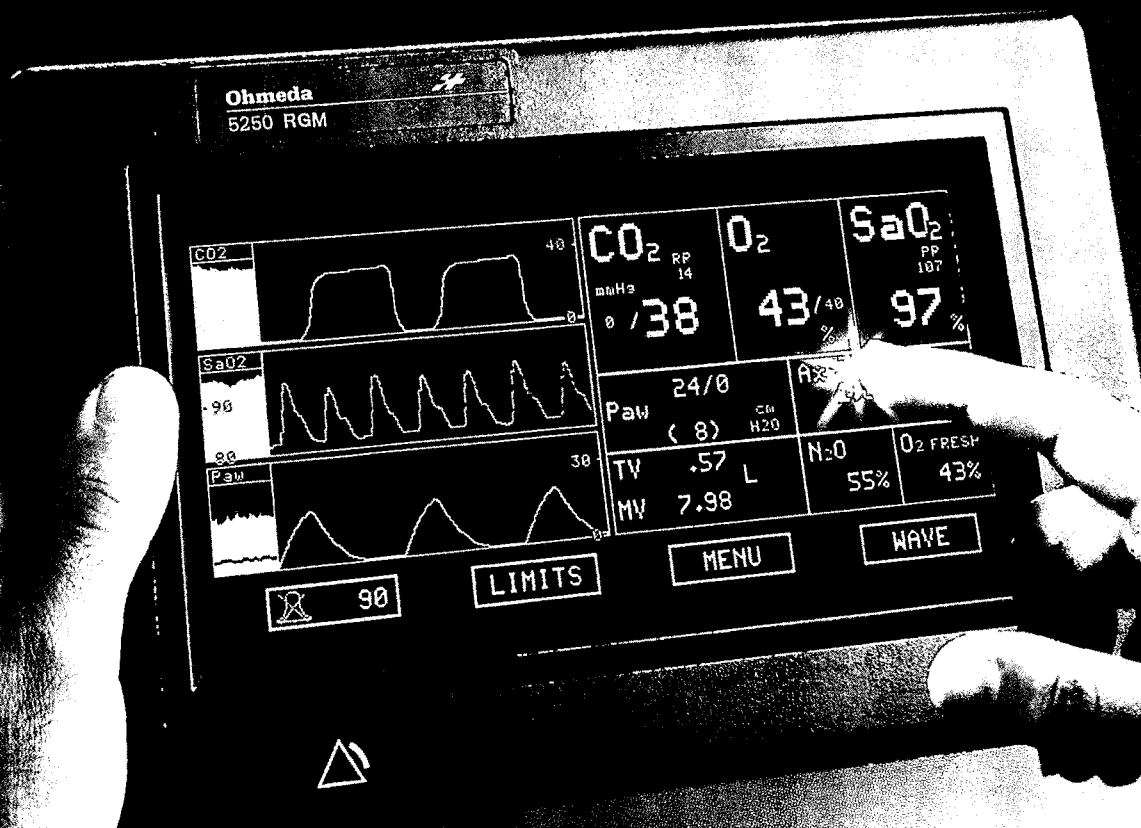
Between Towns Road, Cowley, Oxford, OX4 3LY, England.  
Tel: 0865 748844 Fax: 0865 717313

*Reference*

1. Moore DC. Regional Block. A handbook for use in clinical practice of medicine and surgery. 4th ed. Springfield III: Charles C Thomas; 1964; 358-359.



# When you choose the RGM from Ohmeda, we really have to hand it to you.



...total gas monitoring at your fingertips. Only the Ohmeda 5250 RGM combines up to seven parameters into one flexible system. With its removable touchscreen and easy-to-use menu, it puts the information you need right where you need it.

How does it do it all? We really have to hand it to anesthesiologists like yourself. They wanted a simple, compact alternative to multiple monitors so they could watch digital waveforms and read displays—and their patients.

It's our purpose: the most advanced monitoring technologies in your hands. Just what you'd expect from Ohmeda. Designing products with the clinician's needs in mind for over 80 years.

## Ohmeda

**We monitor life.**

71 Great North Road  
Hatfield  
Herts AL9 5EN  
United Kingdom  
© 0707 263570  
Telex UK 918179

**A BOC Health Care Company**  
Critical Care  
Worldwide

A Division of The BOC Group Inc

© 1990 The BOC Group Inc

system, a 15-litre fresh gas flow and a close fitting mask. Inspired and expired  $O_2$ ,  $N_2O$  and  $CO_2$  were all measured during induction of anaesthesia using an Ohmeda RGM5250 monitor and recorded on a chart recorder alongside the concurrent  $SpO_2$ . Nitrogen washout was considered to be sufficiently complete when the end tidal oxygen was 80% or greater. This was achieved in a mean time of 168 seconds (range 60–260). The mean pre-induction  $SpO_2$  was 97%.

The pre-oxygenated group had a mean age of 50 years (range 18–97,  $n = 20$ ) and had a sex ratio of 12 men to nine women, whilst the non-preoxygenated group had a mean age of 52 years (range 19–82,  $n = 20$ ) and a sex ratio of nine men to 12 women.

In those who were pre-oxygenated the  $SpO_2$  remained little changed at a mean of 98.4% (SD 1.57, range 94–100) whereas in the other group the  $SpO_2$  decreased to a mean of 85.4% (SD 8.49, range 68–95). In this latter group, no patient maintained their  $SpO_2$  and 13 patients had a lowest  $SpO_2$  of 90% or less. In 19 of these the mean time to regain

pre-induction  $SpO_2$  was 78 seconds (SD 73, range 10–300) and in two patients, the  $SpO_2$  never reached pre-induction levels. Two other patients who were not pre-oxygenated before anaesthesia were observed to have an  $SpO_2 < 40\%$  during induction. One had vomited unexpectedly and the other developed laryngospasm. Neither patient was included in the statistical analysis because of the gross distortion of the results so produced. One patient in the pre-oxygenated group developed laryngospasm, but his  $SpO_2$  only decreased to 94%, the lowest observed in that group.

We suggest that serious consideration should be given to the routine use of pre-oxygenation before induction of any form of general anaesthesia, as indeed is commonplace in Australia, Canada and the USA.

University Department of Anaesthesia,  
Royal Liverpool Hospital,  
Liverpool L69 3BX

G. SKEA  
A. JONES  
S.L. SNOWDON

### Fluid balance in ARDS

The results of the questionnaire (*Anaesthesia* 1991; 46: 160–1) on the attitudes of different ICUs towards the problem of fluid balance in the adult respiratory distress syndrome (ARDS) were very interesting. An international panel of experts met in Amsterdam last year in an attempt to reach a consensus on oxygen transport in critical illness and its relevance to patient therapy. All members of the panel agreed that it is better to increase oxygen delivery ( $\dot{V}O_2$ ) with controlled fluid therapy than to limit fluid therapy to 'keep the lungs dry'. It was also agreed that ARDS patients die of organ failure from inadequate  $\dot{V}O_2$  more often than from hypoxia with or without fluid overload. For example, a large study found only 16% of ARDS deaths were due to respiratory failure.<sup>1</sup>

There is now convincing evidence that there is a peripheral defect of oxygen utilisation in ARDS leading to  $\dot{V}O_2$  being dependant on  $\dot{D}O_2$ .<sup>2</sup> Increasing  $\dot{D}O_2$  in patients with ARDS invariably results in an increase in  $\dot{V}O_2$ .<sup>3</sup> Diuretic therapy has not been shown to be effective in reducing lung water in ARDS; this is not surprising. ARDS is an acute inflammation of the lung and diuretics are not designed to reduce inflammation. The danger of overzealous diuretic therapy is renal hypoperfusion. As renal failure is arguably the most important factor in mortality in patients whose lungs are being ventilated, it seems inappropriate to promote its occurrence. Therapy based on oxygen transport is associated, on the other hand, with a reduced incidence of renal failure.<sup>4</sup>

Two studies have addressed the problem of fluid balance in ARDS. The first study<sup>5</sup> showed that there was a relationship between a negative fluid balance and survival but were unable to demonstrate if there was a link between cause and effect or if it was merely another example of sicker patients doing worse. It is of interest to note that the goal of therapy was to achieve the lowest pulmonary capillary wedge pressure (PCWP) consistent with an adequate cardiac output. This is completely different from blind diuretic administration with no haemodynamic measurement. Surely in 1991 there can be no place for management of patients with ARDS without adequate monitoring of therapy, regardless of which school of management you subscribe to.

The second study<sup>6</sup> demonstrated a statistical increase in survival in those patients able to achieve a 25% or greater

decrease in PCWP by a variety of therapeutic interventions. However, this study can be criticised as retrospective and poorly controlled. For example, the survivors were, on average, 18 years younger than the nonsurvivors and could be expected to have a greater cardiac reserve. The editorial accompanying this study concluded that randomised, double-blind, placebo-controlled studies were needed but 'lacking a positive result from this type of prospective study aggressive lowering of pulmonary artery occlusion pressure cannot be currently advocated as a treatment for ARDS'.<sup>7</sup>

Drs McQuillan and Young are correct that further research would be welcome but I believe the current weight of evidence is on the side of optimising preload, cardiac output and  $\dot{D}O_2$ .

The Royal Oldham Hospital,  
Oldham OL1 2JH

I. MCCONACHIE

### References

1. MONTGOMERY AB, STAGER MA, CARRICO J, HUDSON LD. Causes of mortality in patients with the adult respiratory distress syndrome. *American Review of Respiratory Diseases* 1985; 132: 485–9.
2. KARIMAN K, BURNS SR. Regulation of tissue oxygen extraction is disturbed in adult respiratory distress syndrome. *American Review of Respiratory Diseases* 1985; 132: 109–14.
3. MOHSENFAR Z, TASHKIN DP, GOLDBACH P, CAMPISI DJ. Relationship between  $O_2$  delivery and  $O_2$  consumption in the adult respiratory distress syndrome. *Chest* 1983; 84: 267–71.
4. SHOEMAKER WC, APPEL PL, KRAM HB, WAXMAN K, LEE TS. Prospective trial of supranormal values of survivors as therapeutic goals in high risk surgical patients. *Chest* 1988; 94: 1176–86.
5. SIMMONS RS, BERDINE GG, SEIDENFELD JJ, PRIHODA TJ, HARRIS GD, SMITH JD, GILBERT J, MOTA E, JOHANSON WG JNR. Fluid balance and the adult respiratory distress syndrome. *American Review of Respiratory Diseases* 1987; 135: 924–9.
6. HUMPHREY H, HALL J, SZNAJDER I, SILVERSTEIN M, WOOD L. Improved survival in adult respiratory distress syndrome patients associated with a reduction in pulmonary capillary wedge pressure. *Chest* 1990; 97: 1176–80.
7. HYERS TM. ARDS: The therapeutic dilemma. *Chest* 1990; 97: 1025.

## Acupuncture and postoperative sickness

Generalities, while apparently simple, are often incorrect and, if quoted out of context, can be misleading. A good example of this occurs in the interesting paper by Kho and colleagues (*Anaesthesia* 1991; **46**: 129–35) on acupuncture analgesia in major surgery. Although they did not give specific data on nausea and vomiting, their findings suggest that those patients who had acupuncture and transcutaneous stimulation analgesia were less sick (one patient required prochlorperazine) than those having fentanyl alone for analgesia (five required prochlorperazine). This would not be an unexpected finding in view of the different mean doses of fentanyl in the two series (1.2 µg/kg compared with 22.9 µg/kg). In the discussion they state 'Acupuncture has been reported to reduce postoperative nausea' and quote a reference from the Belfast Department in support of this.<sup>1</sup> They attribute the lesser needs for prochlorperazine in patients having acupuncture to this procedure.

While undoubtedly stimulation of the P6 acupuncture point, whether by needling, transcutaneous electrical stimulation, or by pressure has been shown by several groups to have a definite antiemetic action,<sup>2–4</sup> I can find no reference to such an action from stimulation of any of the four body or three ear points used by them. Our own studies in anaesthesia,<sup>2</sup> early pregnancy<sup>5</sup> and with cancer chemotherapy<sup>6</sup> suggest a specificity of action limited to a few points. To attribute this to any acupuncture or transcutaneous stimulation procedure could result in many

patients being disappointed and could lead to an otherwise useful regime falling into disrepute.

Northern Ireland Centre for  
Radiotherapy and Oncology,  
Belvoir Park Hospital,  
Belfast

J.W. DUNDEE

## References

1. DUNDEE JW, CHESTNUTT WN, GHALY RG, LYNAS AGA. Traditional Chinese acupuncture: a potentially useful antiemetic? *British Medical Journal* 1986; **293**: 583–4.
2. DUNDEE JW, GHALY RG, BILL KM, CHESTNUTT WN, FITZPATRICK KTJ, LYNAS AGA. Effect of stimulation of the P6 antiemetic point on postoperative nausea and vomiting. *British Journal of Anaesthesia* 1989; **63**: 612–8.
3. HO RT, JAWAN B, FUNG ST, CHEUNG HK, LEE JH. Electro-acupuncture and postoperative emesis. *Anaesthesia* 1990; **45**: 327–9.
4. BARSOUM G, PERRY EP, FRASER IA. Postoperative nausea is relieved by acupressure. *Journal of the Royal Society of Medicine* 1990; **83**: 86–9.
5. DUNDEE JW, SOURIAL FBR, GHALY RG, BELL PF. P6 acupressure reduces morning sickness. *Journal of the Royal Society of Medicine* 1988; **81**: 456–7.
6. DUNDEE JW, GHALY RG, FITZPATRICK KTJ, ABRAM WP, LYNCH GA. Acupuncture prophylaxis of cancer chemotherapy-induced sickness. *Journal of the Royal Society of Medicine* 1989; **82**: 268–1.

## Failure of hydrocortisone to prevent postoperative sore throat

Dr P.C. Stride (*Anaesthesia* 1990; **45**: 968–70) does a service by telling us that, contrary to expectation, lubrication of tracheal tubes with a steroid cream not only failed to protect against sore throat but actually seemed to make discomfort more likely in comparison with KY jelly. Loeser *et al.*<sup>1</sup> in 600 patients compared no lubrication, Surgilube (a 'bland' lubricant), or 5% lignocaine ointment and found that no lubrication was best and lignocaine the worst. Stock and Downs<sup>2</sup> also examined this problem in 160 patients, comparing no lubrication, normal saline, water-soluble jelly, 2% lignocaine and 2.5% lignocaine ointment; they found no difference.

He referred in his bibliography to a paper by Stout *et al.*<sup>3</sup> but Dr Stride did not mention their most important point, namely that use of a small tube (7 mm instead of 9 mm in men, 6.5 mm instead of 8.5 mm in women) reduced the incidence of sore throat from 48% to 22%. Dr Stride does not say if he controlled for tube size between his two groups.

The common custom of using the biggest tube that the larynx will accept is a heritage from the early days of scientific anaesthesia when it was pointed out by Macintosh and Mushin that resistance to flow through a tube was proportional to the fourth power of its diameter. Thus, if one halved the size of the tube, the resistance to breathing was increased 16-fold. That was important in a patient breathing for himself. But now, when assisted or controlled breathing is the general rule, the increased resistance is harmless since the ventilator does the work. The only effect is a small amount of PEEP.<sup>4</sup>

It's time to change! I have used small unlubricated tubes (6 mm for women, 7 mm for men) for more than 10 years and very rarely does one of my patients complain of sore throat. I recommend that others give this practice a test. Of course, in cardiopulmonary or intracranial cases or those

expected to require postoperative ventilatory support, it is wise to use tubes one size larger.

2500 Hospital Drive No. 10,  
Mountain View  
California 94040  
USA

D.V. THOMAS

## References

1. LOESER EA, KAMINSKY A, DIAZ A, STANLEY TH, PACE NL. The influence of endotracheal cuff design and cuff lubrication on postoperative sore throat. *Anesthesiology* 1983; **58**: 376–9.
2. STOCK MC, DOWNS JB. Lubrication of tracheal tubes to prevent sore throat from intubation. *Anesthesiology* 1982; **57**: 418–20.
3. STOUT DM, BISHOP MJ, DWERSTEG JF, CULLEN BF. Correlation of endotracheal tube size with sore throat and hoarseness following general anesthesia. *Anesthesiology* 1987; **67**: 418–21.
4. STENQVIST O, SONANDER H, NILSSON K. Small endotracheal tubes—ventilator and intratracheal pressures during controlled ventilation. *British Journal of Anaesthesia* 1979; **51**: 375–80.

## A reply

Thank you for the opportunity to reply to Dr Thomas' letter. I am pleased to hear of his success in reducing postoperative sore throat by using small tracheal tubes. As I acknowledged in my paper, Stout *et al.*<sup>1</sup> attributed this finding to a decreased surface area of mucosal contact. I do not understand the observation regarding control of tube size. As I stated, tracheal tube sizes were standardised in both groups of my study (9.0 mm ID for men, 8.0 mm ID for women).



# The World's Premier Medical Publisher

## EPIDURAL & SPINAL BLOCKADE IN OBSTETRICS

Edited by

**Felicity Reynolds**, Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.

This book has evolved from the Obstetric Anaesthetist's Association meeting in the UK in September 1989. Unlike other books of proceedings however, the well-known contributors were invited not only to speak but also to provide a chapter reviewing their subject, prepared especially for the book.

Edited by Dr. Reynolds, the result is an interesting blend of a review of the last ten year's developments coupled with presentations of current research and coloured throughout with stimulating, amusing discussion.

- Includes unique illustrations, using state-of-the-art imaging techniques, of the anatomy of the epidural space.
- Features excellent reviews of the rapidly expanding fields of Caesarean section and the use of spinal opioids.

The closing section, The Recipient, discusses the effects that conduction blocks may have on the baby, and reviews the lay literature covering consumer's views of epidural practice.

### CONTENTS

The Epidural Space: Anatomy and Approach • Indications and Contraindications to Epidural Blockade • Test Doses • Top-ups Versus Continuous Infusion • Management of the Second Stage of Labour • Local Anaesthetic Toxicity • Taps and Patches • Neurological Complications of Epidural and Spinal Blockade • Regional or General Anaesthesia for Emergency Caesarean Section • Spinal and Epidural Blockade • Chairman's Responses • Epidural Opioids in Labour • Epidural Opioids for Caesarean Section • The Effects of Regional Analgesia • Public Opinions • Summary and Conclusions • Index.

**0 7020 1401 X ca 250pp Ills Pb**  
**August 1990 Baillière Tindall £17.50**

## ESSENTIALS OF ANESTHESIOLOGY 2/e

**David C. Chung**, Consultant, Dept Anaesthesiology, The Mississauga Hospital, Ontario, Canada;

**& Arthur M. Lam**, Assoc. Prof., Dept Anaesthesiology, Harborview Medical Center, Univ. Washington, Seattle, USA.

A primer in anaesthesia written for those about to specialise in the field, this book provides concise, yet thorough coverage of the scientific basis and the principles of safe practice. All chapters have been revised to incorporate new drugs, equipment and medical conditions.

Includes:

- Revised and expanded section on management of postoperative pain
- New section on quality assurance and risk management
- New chapter on AIDS and the anaesthetist
- New section on autologous blood transfusions

**0 7216 3084 7 304pp 50 ills SpPb**  
**1989 W.B. Saunders £15.50**

## TEXTBOOK OF PAEDIATRIC ANAESTHETIC PRACTICE

**E. Sumner & D. Hatch**

**0 7020 1336 6 560pp 103 ills Hb**  
**1989 Baillière Tindall £37.50**

## INTENSIVE CARE: A Concise Textbook

**C.J. Hinds**

**0 7020 1150 9 387pp 109 ills Pb**  
**1986 Baillière Tindall £15.95**

## OBSTETRIC ANAESTHESIA & ANALGESIA 3/e

**D.D. Moir & J. Thorburn**

**0 7020 1154 1 390pp Ills Hb**  
**1986 Baillière Tindall £23.00**

## DIFFICULTIES IN TRACHEAL INTUBATION

**I.P. Latta & M. Rosen**

**0 7020 1096 0 192pp 150 ills Hb**  
**1985 Baillière Tindall £19.95**

**W.B. Saunders & Baillière Tindall**

**HARCOURT BRACE JOVANOVIH**

Available from your bookseller. For more information please contact: The Marketing Dept, Harcourt Brace Jovanovich, 24-28 Oval Rd, London NW1 7DX, UK.

OR phone 081 300 3322

Prices and specifications may vary without notice.



Having worked in both the USA and UK, I find it interesting to reflect on the differences in anaesthetic practice between the two countries. While assisted or controlled ventilation is indeed the general rule for intubated patients in the USA, in my experience intubated patients in the UK are *often* left to breathe spontaneously. This may be part of the explanation for the use of larger tubes in the UK. Larger tubes may also be less likely to kink or become obstructed in the presence of copious secretions. With experience, anaesthetists learn to safely extubate patients soon after return of spontaneous ventilation. Inexperienced anaesthetists may be more cautious, especially in the patient with known respiratory disease, such as a chronic bronchitic. In this situation the increased resistance and increased risk of obstruction associated with using the smaller tube may become significant.

Reducing postoperative sore throat (the true incidence of

which may only be revealed by direct and specific questioning)<sup>2</sup> is a desirable aim, but should not be allowed to compromise patient safety. For this reason, I shall continue to teach others what I was myself taught: 'start with a large intravenous cannula ...intubate with a large tube.'

John Sealy Hospital,  
Galveston, Texas 77550,  
USA

P.C. STRIDE

#### References

1. STOUT DM, BISHOP MJ, DWERSTEG JF, CULLEN BF. Correlation of endotracheal tube size with sore throat and hoarseness following general anaesthesia. *Anesthesiology* 1987; **67**: 419-21.
2. HARDING CJ, MCVEY FK. Interview method affects incidence of postoperative sore throat. *Anaesthesia* 1987; **42**: 1104-7.

#### Hypoxaemia during induction of anaesthesia

It was interesting to read the article on arterial oxygen saturation during induction of anaesthesia (*Anaesthesia* 1990, **45**: 1012-5). The practice of pre-oxygenating the patients while inducing anaesthesia is common in Sweden and indeed, barring exceptional circumstances, is a routine for all anaesthetics, whether mask or otherwise. Therefore, it came as a surprise that 87% of anaesthetists in the UK do not do this routinely. The fact that all patients except one who developed hypoxaemia were below 35 years old further highlights the problem in older and relatively unhealthy patients. If the definition of hypoxaemia, on theoretical basis, includes  $SpO_2$  below 94%<sup>1</sup> six patients in the group who had 100%  $O_2$  after loss of eyelash reflex would also be considered to be hypoxaemic. What is even more interesting is that of the nine patients who were considered to be hypoxaemic, only one was male. Is it then possible that females are more likely, or more rapidly, to develop hypoxaemia? In a study done in our hospital some

time ago (unpublished), no patient of 40 years of age undergoing laparoscopy developed hypoxaemia during induction of anaesthesia. We are convinced that this is because of the practise of pre-oxygenation in all patients at the start of induction. The fact that three patients did not get thiopentone for induction (or is it that the dose given is unknown?) came as another surprise in view of the protocol!

Department of Anaesthesiology,  
Faculty of Health Sciences,  
S-58185 Linköping, Sweden

A. GUPTA  
M. VEGFORS  
G. LENNMARKEN

#### Reference

1. TAYLOR MB, WHITWAM JG. The current status of pulse oximetry. *Anaesthesia* 1986; **41**: 943-9.

#### Factors that influence the induction dose of propofol

We were interested to read the study by Drs McLeane, Fogarty and Watters on factors that influence the induction dose of propofol (*Anaesthesia* 1991; **46**: 59-61) but feel that their conclusions are not necessarily valid.

Firstly they comment on the speed of injection possibly affecting the dose requirements. Since their paper was accepted, Peacock *et al.*<sup>1</sup> have shown that the dose requirements diminished with a reduction in the infusion in elderly patients using rates of infusion down to 50 ml/minute. The dose used averaged 1.2 mg/kg/hour, which was less than the dose used by the investigators in this study. Secondly, whilst the authors find a relationship between age and induction dose, we were surprised that they extrapolated their findings of correlations with haemoglobin, albumin and urea whilst making no attempt to correlate these values with the age of the patient, their ASA status or whether the operation was an emergency or not. Stepwise regression analysis may have distinguished the significance of the separate components. Older and sicker patients tend to have a lower albumin and haemoglobin and a higher urea. The authors have not attempted to exclude this effect in their study of apparently unselected patients.

Department of Anaesthesia,  
University of Sheffield Medical School  
Sheffield S10 2RX

G.A. McLAUCHLAN  
P. SPIERS  
J.E. PEACOCK

#### Reference

1. PEACOCK JE, LEWIS RP, REILLY CS, NIMMO WS. Effect of different rates of infusion of propofol for induction of anaesthesia in elderly patients. *British Journal of Anaesthesia* 1990; **66**: 346-52.

#### A reply

Thank you for the opportunity to reply to this letter. We are pleased that Dr McLauchlan and colleagues have confirmed our clinical impression that the speed of injection of propofol influences the dose required to achieve induction. The influence of age on the induction dose is interesting. Age is a crude estimate of the degenerative processes that inevitably occur during life. With advancing age, the incidence of disease (and hence abnormal haematological and biochemical results) increases. However, patients with a relatively low chronological age

may still have advanced disease states that radically alter the normal physiological and biochemical mechanisms of the body and it would not be surprising if the induction dose of an agent were altered in these patients. It may well be that the relationship between age and the required induction dose of propofol is a consequence of abnormal haemoglobin, albumin and urea concentrations, which occur more frequently in these patients rather than an effect of age as such. Furthermore, by definition, abnormal concentrations of haemoglobin, urea and albumin do not

occur in health and hence patients in this study who have abnormal levels of these variables were 'sick' (and some, but not all, were more elderly). Therefore we feel it inappropriate to differentiate between 'sick' patients and those with abnormal biochemical and haematological results.

Department of Anaesthetics,  
Craigavon Area Hospital,  
Craigavon, Co Armagh

G.J. MCCLEANE

### Hyoscine derivative in children—but was it hyoscine?

I was interested to read the letter by Dr Danks (*Anaesthesia* 1990; 45: 1089) concerning hyoscine overdose in a child. Adherence to the simple rule that only anaesthetists should administer drugs to an anaesthetised patient would have avoided this problem. However, this report contains important implications; the most notable was, was it in fact the hyoscine that caused the problems?

Firstly, undesirable central (i.e. behavioural) side effects of anticholinergic drugs are common, although unpredictable in severity. Anaesthetists must be aware of the signs of the central anticholinergic syndrome, since it may be encountered postoperatively<sup>1</sup> or following drug overdose. Specific treatment is by slow intravenous injection of physostigmine 0.04 mg/kg, one dose usually being sufficient in postoperative cases. Physostigmine should always be available in all areas where anaesthetics are given, in psychiatric wards and in accident and emergency units. Besides its use for treatment of the anticholinergic syndrome, it can also be given to aid in the differential diagnosis of drug overdose where sedation is a feature. It is a pity that physostigmine was not given to Dr Danks' patient, as reversal of sedation would have indicated that the delayed recovery was indeed an anticholinergic effect.

Secondly, it is doubtful that hyoscine-N-butyl bromide was the cause, since it is a quaternary derivative of hyoscine<sup>2</sup> and therefore unlikely to have any central actions. The manufacturers (Boehringer-Ingelheim) were unable to provide me with any report of any central effects of an intravenous overdose of Buscopan. Furthermore, it has been shown in cats that a large overdose of quaternary atropine caused no EEG changes, which is in marked

contrast to the profound EEG doses following small amounts of atropine sulphate.<sup>3</sup>

Thirdly, therefore, it would seem reasonable to seek a completely different explanation for the postoperative problems found in Dr Danks' patient, unless it can be shown that Buscopan can penetrate the blood brain barrier in the presence of mild jaundice in an otherwise fit child. Did the child have an unknown medical condition or was the anaesthetic to blame?

It must always be borne in mind that quaternary compounds do not act centrally in normal individuals, but also that nonquaternary anticholinergic agents can have adverse central effects. Although the latter can be reversed with physostigmine, they can be avoided altogether by use of glycopyrronium or atropine methyl nitrate.

Erasmus University  
University Hospital Dijkzigt  
Rotterdam, The Netherlands

J. RUPREHT

### References

1. RUPREHT J, DWORACEK B. The central anticholinergic syndrome in the postoperative period. In: NUNN JF, UTTING JE, BROWN BR Jr, eds. *General anaesthesia*, 5th edn. London: Butterworths, 1989. 114–8.
2. Atropine and other anticholinergic agents. In: REYNOLDS JEF ed. *Martindale, The Extra Pharmacopoeia* 28th ed, London: The Pharmaceutical Press, 1982: 302.
3. RUPREHT J, DWORACEK B, DUCARDUS R. Electrocardiographic changes after intoxication with anticholinergic drugs. *Acta Anaesthesiologica Belgica* 1983; 34: 123–9.

### Emergency adenotonsillectomy for acute airway obstruction

The case report by Livesey *et al.* (*Anaesthesia* 1991; 46: 36–7) raises a number of important issues with regard to paediatric patients with airway obstruction.

Analgesia was provided with intravenous papaveretum. Studies have shown that regional anaesthesia is superior to parenteral opioids in relieving pain postoperatively.<sup>1,2</sup> Opioids have been shown to predispose to oxygen desaturation in the postoperative period<sup>3</sup> and an opioid-sparing effect is useful if there is any degree of respiratory obstruction. No mention is made of whether opioids were given postoperatively, a factor which may have precipitated the cyanotic incident reported. Regional anaesthesia should be considered in these patients. It is also safe practice to monitor patients with any degree of airway obstruction with pulse oximetry and an apnoea mattress.

The incidence of tonsillar enlargement is high in the paediatric population and anaesthetists should be aware of this fact when managing these patients for unrelated

conditions. The authors rightly caution against the use of respiratory depressant premedicants in any patient with potential airway obstruction.

St. George's Hospital,  
London SW17 0QT

G. FRANCIS

### References

1. MCGOWN RG. Caudal analgesia in children: Five hundred cases for procedures below the diaphragm. *Anaesthesia* 1982; 37: 806–18.
2. LUNN JN. Postoperative analgesia after circumcision: A randomised comparison between caudal analgesia and intramuscular morphine in boys. *Anaesthesia* 1979; 34: 552–4.
3. CATLEY DM, THORNTON C, JORDAN C, LEHANE JR, ROYSTON D, JONE JG. Pronounced episodic oxygen desaturation in the postoperative period: Its association with ventilatory pattern and analgesia regimen. *Anesthesiology* 1985; 63: 20–8.



*A reply*

Opioids were only given intra-operatively and not postoperatively in our patient. Regional anaesthesia is commonly used in our hospital for children undergoing operations such as orchidopexy, but was not used in this case. I would agree with Dr Francis on its value, particularly in patients with any degree of upper airway obstruction. With regard to the question of pulse oximetry, this is used routinely for monitoring purposes. Once again the emphasis must be on awareness of the potential risks and problems as stated in the original paper.

ENT Department,  
Royal Surrey County Hospital,  
Guildford GU2 5XX

N. SOLOMONS

We were interested to read the case report by Drs Livesey, Solomons and Gillies (*Anaesthesia* 1991; 46: 36-7) on acute upper airway obstruction in a child with severe adeno tonsillar hypertrophy, as we have encountered a similar case. Acute tonsillitis is a very rare cause of progressive upper airway obstruction.<sup>1</sup> A one-year-old African boy with sickle cell trait was seen in the ENT outpatient clinic with a 6-month history of mouth breathing, loud stertor and recurrent upper respiratory tract infections. Examination revealed nasal obstruction with acute follicular tonsillitis. A 2-week course of oral erythromycin and ephedrine nasal drops was prescribed.

During the next 2 weeks a cough developed, the stertor increased and sporadic apnoeic spells occurred during sleep, interrupted by restless awakening. A deterioration in symptoms led to emergency admission at night. Whilst the child was asleep, inspiratory stridor was present associated with tracheal tug, intercostal recession and frequent periods of obstructive apnoea of 15 seconds or longer. The stridor and airway obstruction improved markedly on awakening. He was pyrexial (39°C). However, there was no drooling, he did not favour the sitting position and had readily been able to drink fluids at home. These features, together with the slow onset and history, made acute tonsillitis leading to upper airway obstruction the most likely diagnosis rather than epiglottitis. No attempt was made to disturb the child, and he was transferred direct to the operating theatre for laryngoscopy and was anaesthetised by inhalational induction using halothane in oxygen. During induction, complete airway obstruction occurred, which was only relieved by the use of a Guedel airway. Laryngoscopy revealed large infected tonsils meeting in the midline with an inflamed oropharynx. There was no evidence of a

peritonsillar or retropharyngeal abscess; the epiglottis and larynx were normal. Oral tracheal intubation was effected without difficulty; examination of the postnasal space confirmed adenoidal hypertrophy. An immediate adenotonsillectomy was contra-indicated due to the risk of excessive haemorrhage resulting from the inflammation.<sup>2</sup> The oral tracheal tube was changed to the nasal route and the child kept sedated in the intensive care unit. He was treated with intravenous cefuroxime and an adenotonsillectomy was performed 48 hours later.

The tube was kept in place for a further 24 hours allowing recovery from sedation and enhanced safety in the event of postoperative haemorrhage. Moreover, a recent report has indicated that adenotonsillectomy in young children has increased the risk of postoperative airway obstruction where pre-operative airway obstruction existed.<sup>3</sup> Apart from mild postextubation stridor there were no complications. He was discharged home 7 days later with complete resolution of his symptoms.

Whilst adenotonsillar enlargement is a recognised cause of obstructive sleep apnoea in children,<sup>4</sup> progressive airway obstruction may occur in acute tonsillitis, but is very rare. The clinical features in this case distinguished it from acute epiglottitis. The initial management was similar to that recommended for acute epiglottitis,<sup>5</sup> with the exception of the Guedel airway insertion which presumably acted by parting the tonsils. Adenotonsillectomy is rarely indicated under the age of 4 years and very rarely under 18 months; acute upper airway obstruction, however, is one of the few indications for tonsillectomy at any age.

Papworth Hospital,  
Cambridge CB3 8RE

R.J. HARWOOD  
T.A. KING  
D.F. JOHNSTON  
J.C. WATKINSON

*References*

1. HIBBERT J. Acute infection of the Pharynx and Tonsils. In: KERR AG, GROVES J, STELL PM, eds. *Scott Brown's Otolaryngology*, 5th edn. London: Butterworths, 1987; 5: 76-98.
2. ZALZAL GH, COTTON RT. Adeno-tonsillar Disease. In: CUMMINGS CW, SCHULLER DE, eds. *Otolaryngology—Head and neck surgery*. St. Louis, C.V. Mosby 1986; 2: 1189-211.
3. BERKOWITZ RG, ZALZAL GH. Tonsillectomy in children under three years of age. *Archives of Otolaryngology—Head and neck surgery*. 1990; 116: 685-6.
4. GOODE RL. Sleep Disorders. In: CUMMINGS CW, SCHULLER DE, eds. *Otolaryngology—Head and neck surgery*. St. Louis: C.V. Mosby, 1986; 2: 1449-1457.
5. JAMES I. EPIGLOTTITIS. *Intensive and Critical Care Digest*. 1987; 6: 40-1.

**Does complicated mean better?**

The classic Boyle's anaesthetic machine is swiftly changing, but not for the better. I have recently met, and used, several 'state-of-the-art' trolleys which are best described as modern (and expensive) dinosaurs. They certainly frighten me! Gone is the simplicity and elegance of the old machines. Now we have these large tanks with bulky closed circuitry, a high column of electronic devices, an intimidating and inflexible flowmeter system (conforming, I am told, with Department of Health Requirements ... the carbon dioxide gang strikes again?), an elaborate ventilator with all sorts of knobs, ill-designed vaporizers whose slots will accept no others ... one hospital was offered such a trolley for £3000 less than the manufacturer's

recommended price. A hopeful sign of sales resistance? Do these trolleys reflect the fruitless aim of trying to please everyone? They remind me of today's ventilators, very complicated and very expensive, although without any scientific evidence that they favourably influence the course of acute pulmonary failure. Elegance and simplicity should be primary goals in equipment design unless both ugliness and complexity are of proven value. As to ergonomics, may be that's too much to expect.

20 Hocroft Avenue,  
London NW2 2EH

A. GILSTON

## Faulty portex epidural needle

I would like to report a fault in the manufacture and/or quality control of a Portex Tuohy needle (lot no. 90E 02). The position of the notch into which the stylet locks in the orifice should be in the same direction in which the bevel faces. In this particular situation, the bevel points in the



Fig. 1. Epidural needle with stylet lock and bevel in opposite directions.

opposite direction, i.e., downwards (Fig. 1). This resulted in a degree of confusion for the anaesthetist.

Mannerheimintie 38 A 2a  
00100 Helsinki,  
Finland

D.A. COZANITIS

## A reply

Portex is a responsible manufacturer of single-use medical devices and as such applies rigorous controls to product design, manufacture and quality. Our manufacturing methods have been designed to avoid the risk of misorientation of components assembled or moulded together. This is the first report we have received of a Tuohy needle with the hub 180° out of alignment with the bevel.

We have reviewed our manufacturing systems and have further enhanced the methods by which we positively orientate the needle prior to moulding on of the hub.

Notwithstanding the strict controls which we apply to design, manufacture and quality assurance, we would commend the standard practice of checking equipment prior to use.

Group Marketing Director,  
Portex Ltd,  
Hythe,  
Kent CT21 6JL

N. GREEN

## A retrospective analysis of epidural vein trauma

Two recent articles (*Anaesthesia* 1989; 45: 788 and 920–3) have both concluded that the risk of blood vessel trauma during epidural catheter insertion is irrespective of whether the patient is in the lateral or sitting position. Prior injection of a bolus of fluid through the epidural needle has, however, been shown to decrease the incidence of blood vessel puncture after catheter placement in laterally positioned patients.<sup>1</sup>

Details concerning epidural catheter placement, including positioning of the patient, together with the technique used to identify loss of resistance, were analysed retrospectively over an 18-month period at the Birmingham Maternity Hospital. The epidural space was located by loss of resistance either to air or saline. In the latter case it was presumed that a bolus of fluid was injected into the epidural space before catheter insertion. Where loss of resistance to air was followed by a bolus of fluid prior to catheter insertion, the technique was identified as loss of resistance to saline for the purposes of this review. Otherwise, the usual method following identification of the epidural space by air was catheter insertion, without prior injection of fluid. The positioning of the patient for the epidural catheterisation was also noted. During this time, epidural catheters were inserted in 909 mothers. The overall incidence of epidural vein trauma during catheter insertion was 11.1%.

Disregarding positioning of the patient, when the technique used for loss of resistance was with saline, blood was seen in 31 patients out of 259 (13%). When air was used blood was seen in the catheter in 70 out of 650 patients (10.7%). This was not statistically significantly different. When the loss of resistance technique was disregarded, blood vessel puncture occurred in 41 of 368 mothers (11.1%) in the sitting position, compared to 60 of 541 (11.1%) in the lateral. When the technique used was only loss of resistance to air, the position of the patient again had no significant effect on the incidence of blood vessel puncture with the catheter (24 of 227 sitting, 48% 423 lateral). With the saline technique the corresponding figures were 17% 141 and 14% 118.

The injection of a bolus of fluid into the epidural space before epidural catheter insertion is advocated on the basis

that this will displace epidural veins out of the way of the following catheter.<sup>2</sup> This retrospective analysis does not support this view. However, it is possible that the amount of fluid injected in the patients reported here (1.0–5.0 ml) was not enough to displace the epidural veins. The practice of injecting fluid before catheter insertion has, however<sup>3</sup> been questioned, on the basis that any fluid then aspirated via the catheter is difficult to identify in that it might be cerebrospinal fluid.

Positioning of the patient is also believed to help in reducing the incidence of epidural vein trauma. The veins in the lumbar region are engaged, so it is believed that there is an increased risk of vessel puncture. The present retrospective analysis does not support this view and would seem to come to the same conclusions as the two articles referred to earlier. In particular, it supports the conclusion of Stone and colleagues that it is probably the skill and experience of the anaesthetist in keeping to the midline, where the plexus of epidural veins is believed to be thinnest,<sup>2</sup> that is the major factor influencing the incidence of vein trauma.

Cannulation of an epidural vein on catheter insertion is a major inconvenience. If undetected there is the danger of intravenous injection of local anaesthetic which can have serious consequences.<sup>3</sup> This retrospective analysis on the practices of a group of anaesthetists, of varying grades and experience, would seem to show that there is no effective prophylactic measure to prevent blood vessel puncture with an epidural catheter and further supports the need for test doses prior to catheter top-ups.

Birmingham Maternity Hospital,  
Birmingham,  
West Midlands

A.L. SKIDMORE

## References

1. VERNIQUET AJW. Vessel puncture with epidural catheters *Anaesthesia* 1980; 35: 660–2.
2. BROMAGE PR. Epidural analgesia. Philadelphia: W.B. Saunders 1978.
3. MARX GF. Cardiotoxicity of local anesthetics—the plot thickens. *Anesthesiology* 1984; 60: 3–5.

### Three in one block for fractured neck of femur

I read with interest the paper of Hood, Edbrooke, and Gerrish, (*Anaesthesia* 1990; 46: 138–40) since I have been using this technique for some years. As they say, the extent of postoperative analgesia can be extended by using bupivacaine instead of prilocaine. However the versatility of the block can be improved by using an indwelling cannula.<sup>1</sup> If this is inserted on admission, boluses of local anaesthetic for pre-operative analgesia may be administered as required. This technique can be used to cover painful manoeuvres such as turning the patient in bed and positioning for subarachnoid block.

I conducted a small uncontrolled study to assess the feasibility of continuous infusion of local anaesthetic through an indwelling cannula in 15 patients who were having either compression screw fixation of fractured neck of femur or hemi-arthroplasty. An 18-G cannula was inserted beneath the inguinal ligament into the femoral 'sheath' and an extension tube was attached to facilitate administration of local anaesthetic. A bolus of bupivacaine was given to provide analgesia during general anaesthesia or for positioning the patient for subarachnoid block. A continuous infusion of 0.25% bupivacaine was then started at 10 ml/hour and continued until the patient started mobilising after the operation. The longest period of infusion was 72 hours. Good analgesia was achieved in every case. No adverse effects were seen. In particular there were no signs of systemic toxicity and no problems with mobilisation due to motor block. There were no cases of local sepsis associated with the cannula.

One case history illustrates the value of the block. An 82-year-old ex-miner had a 20 year history of pneumoconiosis with severe respiratory impairment. Shortly after admission with a fractured neck of femur he suffered a respiratory arrest after 10 mg of intramuscular papaveretum, which was treated with naloxone. Analgesia was later provided with a three in one block. After operation he returned to the ward where he became profoundly cyanosed after accidental discontinuation of oxygen therapy. He was admitted to the Intensive Therapy Unit where a continuous three in one block facilitated his management without opioids; he made an uneventful recovery.

This study also showed that the three in one block is satisfactory for postoperative analgesia in hemi-arthroplasty in spite of the theoretical objections raised by Hood and co-workers. However, I support the conclusions of these workers and suggest that continuous infusion or bolus top-ups of local anaesthetic via a femoral cannula is safe and extends the applications of the three in one.

44, Berwyn Avenue,  
Thingwall  
Wirral L61 7UW

A.G. JONES

### Reference

1. POSTEL J, MÄRZ P. Die Kontinuierliche Blockade des Plexus lumbalis ("3 in 1" block) in der Perioperativen Schmerztherapie. *Regional-anaesthesia* 1984; 7: 140–143.

### Dilatation of hand veins

Drs Williams and Hecker (*Anaesthesia* 1991; 46: 14–6) have described a method of dilating hand veins before venepuncture and providing analgesia by the application of ice. I would like to suggest my own method of dilating hand and wrist veins. I always wear a pair of surgical gloves in the anaesthetic room and operating theatre and discovered that my hand veins always dilate. Whenever I have a patient where venepuncture may be difficult, I give

them gloves to wear. Within 5 minutes the hands are warm and dilated. I always use local infiltration for intravenous cannulations but in future may use ice to provide an alternative form of analgesia.

Whittington Hospital  
London N19 5NF

D.V.A. KALMANOVITCH

### Pulse oximetry in the postcardiopulmonary bypass period

It is interesting to compare the paper by Clayton *et al.* (*Anaesthesia* 1991; 46: 3–10) with our recent work.<sup>1</sup> Both studies examined patients recovering from cardiopulmonary bypass (CPB). The Australian study compared the performance of 20 different pulse oximeters at one point shortly after the end of surgery, examining their accuracy, precision and 95% confidence limits. We evaluated one type of pulse oximeter (Ohmeda Biox 3700) sequentially throughout the weaning period.

The Australian authors found only two of the 20 pulse oximeters tested gave 95% of results within 4% of that obtained with the 'gold standard' co-oximeter. The Ohmeda Biox 3700 gave a poor performance, ranking 14th on accuracy, 18th on number of readings within 3% of the co-oximeter and 20th for positive limit. The mean accuracy was 1.3% with a mean recorded temperature of  $35.1 \pm 0.75^\circ\text{C}$ , which compares well to our figure of 1.5% at  $< 35^\circ\text{C}$ , although our oximeter readings were compared to arterial oxygen saturation calculated from blood gas results (IL 1312). We found that the mean accuracy was between 1

and 2% at all temperature ranges studied and was unaffected by poor perfusion as assessed by systemic vascular resistance.

We also examined clinical performance by determining the number of episodes of reduced saturation ( $< 95\%$ ) detected by the oximeter. This was only 50%, although there were no instances of saturation  $< 90\%$ , so we were unable to comment on accuracy below this level. The failures to detect low saturation were not related to low cardiac output or high systemic vascular resistance. We were unable to obtain an oximeter reading in only three cases (5%). This compared with the Australian study in which the failure rate was 10% for the Ohmeda 3700, with an overall failure rate of 8.7%.

A further point of interest is the carboxyhaemoglobin (HbCO) level quoted by Clayton *et al.* of 2.0–2.1%. It has been suggested in a previous study<sup>2</sup> that, since pulse oximeters are unable to distinguish between carboxy- and oxyhaemoglobin, some part of the overestimation of oxygen saturation by pulse oximetry may be due to the

HbCO concentration. The 2.0% level quoted could more than account for 14 of the 16 overestimation errors reported. It would be interesting to look at this effect more closely comparing smokers and non-smokers.

*St. Thomas' Hospital,  
London SE1 7EH*

D.E. WITHINGTON

#### References

1. WITHINGTON DE, RAMSAY JG, TAZI SAUD A, BILODEAU J. Weaning from ventilation post-cardiopulmonary bypass: evaluation of a non-invasive technique. *Canadian Journal of Anaesthesia* 1991; **38**: 15-9.
2. PALVE H, VOURI A. Pulse oximetry during low cardiac output and hypothermia states immediately after open heart surgery. *Critical Care Medicine* 1989; **17**: 66-9.

#### Anaesthesia for cataract surgery

I fail to see how different forms of anaesthesia affect operating times or waiting lists for cataract surgery. Our operating lists are made up for the number of operations the surgeon can complete in the allotted time. Then, and only then, is the form of anaesthesia decided upon. In the absence of contraindications to local or general anaesthesia, the patient is free to choose between them. If an elderly person is able to go home shortly after general anaesthesia for surgery to their foot, they can surely do so following eye surgery. The operating time is the limiting

factor. By all means decide to perform all cataract surgery under local anaesthesia, but do not be under any illusion that there is any other reason than cost containment. A surprising number of elderly people, given a fully free choice, will elect to have a general anaesthetic, and this will not influence the length of their postoperative stay.

*Queen Elizabeth II Hospital,  
Welwyn Garden City*

J.E. GOOLD

#### An alternative throat spray

The convenience of the xylocaine spray (Astra) can be considerably improved by adding an extension to the spray nozzle. A 14G Angiocath 3.25-inch cannula (Deseret, Beckton Dickinson) can be attached with ease, (Fig. 1) enabling the larynx to be reached. The 14G Abbocath-T 5-5-inch in (Abbott) is longer, fits equally well and can be advanced into the trachea using fingers while the spray bottle is held in the same hand. This arrangement is a useful alternative to the familiar Macintosh or Forrester designs.

*Royal West Sussex Hospital  
Chichester, PO19 4SE*

M.R. NOTT

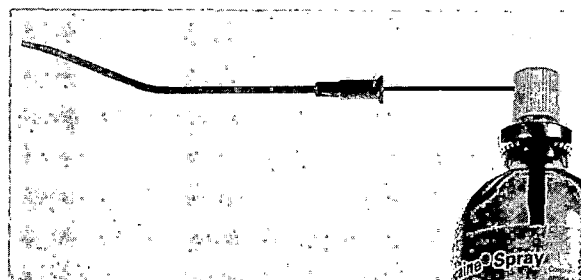


Fig. 1. Alternative throat spray.

#### Withdrawal of levorphanol

I was pleased to read the letter on this matter by R.H. James (*Anaesthesia* 1991; **46**: 71-2). He is not alone in his dismay at the withdrawal of drugs which have a valued place in our armamentarium and yet have ceased to become profitable because they are no longer prominent in current literature. Levorphanol was not recently a widely used drug and yet in my experience it filled a very individual role in the management of chronic pain. There are always a few patients who are unable to tolerate morphine, often because of nonresolving sedative or dysphoric responses and yet the nature of their pain, being predominantly nociceptive, whether it resulted from a malignant condition or severe musculoskeletal problems, indicates that an opioid should be effective. In such circumstances it has always been useful to have a range of alternative longer acting opioids available from which to select. Levorphanol proved extremely useful in this context, especially in the elderly, where it was generally well tolerated and appreciated for its duration of response.

I was currently managing several elderly patients who

had been stabilised on levorphanol when it was withdrawn. It was chosen as a drug which the patient easily tolerated and was found to be the only effective means of controlling pain. I have been unable to find an alternative opioid medication for some of these patients which they find to be as acceptable.

Experience with drug withdrawals in recent years has taught us to expect these decisions to be without consultation with the consumer and often with no prior warning. Complaining to the pharmaceutical industry has in the past had no effect and so I am afraid we accept the commercial decisions meekly. We cannot really blame drug companies who have to ensure that their enterprises are profitable. Perhaps we are partly to blame for not continuing to sing the praises of some long established drugs and convincing our colleagues that some still deserve attention despite the seductive charms of newcomers.

*Frenchay Hospital,  
Bristol BS16 1LE*

S.W. CONIAM

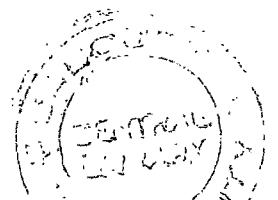
**Dr Gordon Knowles**

As I was responsible for writing Dr Gordon Knowles' Obituary, an edited version of which was published in the *British Medical Journal* (BMJ 1990; 301: 384), may I be permitted to correct a correction (*Anaesthesia* 1991; 46: 164). Dr Knowles qualified MRCS, LRCP in 1937 and MBBS in 1938 from the London Hospital Medical School and not from Leeds. My compliments to Dr David Zuck

for reminding us of Dr Knowles' classification of patients for prostatectomy and that he was inaugural Chairman of the North London Group of Anaesthetists.

*North Middlesex Hospital,  
London N18 1QX*

A.K. MATHUR



## **Safety Action Bulletin**

### **Recovery room trolley: entrapment of patient's fingers between the head section and top framework SAB(91)3**

A patient attempted to lift his upper trunk on an AC Daniels Welwyn and Hatfield Recovery Room Trolley with the head section in the first elevated position from the horizontal. The head section 'snapped' into the flat position, trapping his fingers. The manufacturer (Alpha Trevillton) has now ceased trading so any modification should be carried out by a competent engineer.

### **Stretcher canvases/sheets: faulty seams SAB(91)4**

Some new polyester/cotton stretcher canvases have been found with faulty seams at the pole support casings, which causes the stitching to come apart. The fault is only apparent after they have been laundered.

# Baillière's International Practice and Research CLINICAL ANAESTHESIOLOGY

## Editorial Board

A.R. Aitkenhead (U.K.), M.J. Cousins (Australia), J.F. Crul (The Netherlands), J.G. Jones (U.K.),  
J.F. Nunn (U.K.), L. Strunin (Canada), D. Thomson (Switzerland), H. Van Aken (Belgium)

*Baillière's Clinical Anaesthesiology* is the essential reference for the busy clinician. Each issue is devoted to a separate topic and provides comprehensive coverage from experts in the field. Containing up-to-the-moment reviews, this international reference provides an invaluable source of current clinical opinion, procedures and techniques in anaesthesiology, supported by relevant research data.

## THE 1991 ISSUES

### No. 1 INFECTION AND THE ANAESTHETIST

Guest Editors:  
C.P. Stoutenbeck (Holland)  
& H.K.F. Van Saene (UK)

### No. 2 INTRAVENOUS ANAESTHESIOLOGY - WHAT IS NEW?

Guest Editors: J.W. Dundee (N.Ireland)  
& J.W. Sear (UK)

### No. 3 KINETICS OF ANAESTHETIC DRUGS IN CLINICAL ANAESTHESIOLOGY

Guest Editor: P.F. White (USA)

## PREVIOUS ISSUE TITLES TO COMPLETE YOUR REFERENCE

### Volume 4, 1990

No. 1  
AUTOMATED ANAESTHETIC RECORDS  
Guest Editor: G. Kenny (U.K.)

No. 2  
INTENSIVE CARE:  
DEVELOPMENTS AND CONTROVERSIES  
Guest Editor: G. Dobb (Australia)

No. 3  
ANAESTHESIA FOR DAY CARE SURGERY  
Guest Editor: T.E.J. Healy (U.K.)

### Volume 3, 1989

No. 1  
ANAESTHESIA FOR THE COMPROMISED HEART  
Guest Editor: P. Foex (U.K.)

No. 2  
METABOLIC RESPONSE TO SURGERY  
Guest Editor: D.R. Bevan (Canada)

No. 3  
DEPTH OF ANAESTHESIA  
Guest Editor: J.G. Jones (U.K.)

## ACKNOWLEDGED BY THE MEDICAL PRESS...

"A plethora of clinically applicable facts and therapy"..."An excellent source of reference for all clinicians"..."Up to date"..."Well written"..."Practical"..."Well produced and illustrated"..."Readable"..."Well balanced"..."Excellent source of reference"..."Concise"..."Comprehensive references"..."Excellent value"..."Cost effective"...

Publication: Volume 5 1991 (3 issues)

Subscription Rate: £41.00 (U.K. and Eire)/£47.00 (All other areas). Single Issues: £27.50 (All areas)



Published by Baillière Tindall  
Harcourt Brace Jovanovich, Publishers



## ORDER FORM: Baillière's Clinical Anaesthesiology

Please return to: Journals Marketing Dept., Harcourt Brace Jovanovich Ltd.,  
24-28 Oval Road, London NW1 7DX, U.K. Tel: 071-267-4466

ISSN: 0950-3501

Publication: Volume 5, 1991 (3 issues)

Prepayment is required

☐ Please enter my subscription for Volume 5, 1991

Back volumes and issues are available  
at current year prices.

☐ Please send me \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

### PRICES:

Annual Subscription: £41.00 (U.K. and Eire)/£47.00 (All other areas).

Single Issues: £27.50 (All areas)

☐ I enclose a cheque for £.....  
(made payable to Harcourt Brace Jovanovich Ltd.)

☐ I wish to pay by credit card Amount £.....  
Diners Club / American Express / Visa (Barclaycard) /  
MasterCard (please delete as appropriate)

Card No. \_\_\_\_\_

MasterCard users should add the numbers appearing above their name

Expiry Date \_\_\_\_\_

Signature \_\_\_\_\_

(Your signature is essential, especially when ordering by credit card)

Name \_\_\_\_\_

Address \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

1991 prices are valid until December 31st 1991

Date

JMD/0291/A789



# Contents: Anaesthesia, vol. 46, no. 6, June 1991

## EDITORIAL

### Purchasing equipment

*J. Lumley and D.A. Zideman*

433

## ORIGINAL ARTICLES

### Incidence of awareness with recall during general anaesthesia

*W.H.D. Liu, T.A.S. Thorp, S.G. Graham and A.R. Aitkenhead*

435

### Patient-controlled on-demand epidural fentanyl. A comparison of patient-controlled on-demand fentanyl delivered epidurally or intravenously

*E.A. Welchew and D.P. Breen*

438

### A comparison of APACHE II and a clinical sickness score. A study of 97 consecutive admissions to a District General Hospital Intensive Care Unit

*J.R. Sinclair, P.T. Magee, T.H. Gould and C.H. Collins*

442

### Comparison of extradural and intravenous diamorphine as a supplement to extradural bupivacaine

*A. Lee, D. McKeown, M. Brockway, J. Bannister and J.A.W. Wildsmith*

447

### Propofol in short gynaecological procedures. Comparison of recovery over 2 days after anaesthesia with propofol or thiopentone as sole anaesthetic agent

*L.D. Sanders, P.A. Clyburn, M. Rosen and J.O. Robinson*

451

### Peri-operative drug prescribing pattern and manufacturers' guidelines. An audit

*M.T. Khuger, S. Gale, J.L. Plummer and H. Owen*

456

### Doxapram after general anaesthesia. Its role in stopping shivering during recovery

*V. Sarma and E.N.S. Fry*

460

## CASE REPORTS

### Cardiac anaesthesia in a patient with myotonic dystrophy

*M. Tanaka and Y. Tanaka*

462

### Vocal cord paralysis in the Shy-Drager syndrome. A cause of postoperative respiratory obstruction

*P.M.E. Drury and E.G.N. Williams*

466

### Anaesthesia for a patient with giant axonal neuropathy

*A. Mitchell and P.E. Moskovits*

469

### Resedation after bolus administration of midazolam to an infant and its reversal by flumazenil

*S. Collins and J.A. Carter*

471

### Myoclonic spasms after epidural diamorphine infusion

*B. Jayawardena and D.J. Hill*

473

## APPARATUS

### Minitracheotomy Seldinger—assessment of a new technique

*I.J.B. Jackson, A.K. Choudhry, D.W. Ryan, H.R. Matthews and C.F. Corke*

475

### Evaluation of the Lamtec anaesthetic agent monitor

*S.J.E. Humphrey, N.P. Luff and D.C. White*

478

### Evaluation of the Graseby PCAS. A clinical and laboratory study

*I.J.B. Jackson, P. Semple and J.D. Stevens*

482

## SPECIAL ARTICLE

### Occupational health and pollution from anaesthetics. A report of a seminar

*M.J. Halsey*

486

## FORUM

### The laryngeal mask airway in children. A fiberoptic assessment of positioning

*S.J. Rowbottom, D.L. Simpson and D. Grubb*

489

### Visual evoked potentials and visual acuity after transurethral resection of the prostate

*S. Mantha, S.M. Rao, A.K. Singh, S. Mohandas, B.S.P. Rao and N. Joshi*

491

### End-tidal CO<sub>2</sub> monitoring in mitral stenosis patients undergoing closed mitral commissurotomy

*G.D. Puri, R. Venkatraman and H. Singh*

494

### Antagonism of atracurium with neostigmine. Effect of dose on speed of recovery

*D.R. Goldhill, J.A. Carter, D. Suresh, J.P. Whitehead and P.J. Flynn*

496

### Serum bupivacaine concentrations following wound infiltration in children undergoing inguinal herniotomy

*K.A. Mobley, J.G. Wandless, D. Fell and A. McBurney*

500

## CORRESPONDENCE

502

## SAFETY ACTION BULLETIN

520



VCM

# Anaesth

Journal of the Association of Anaesthetists of Great Britain and Ireland

Volume 46 Number 7 July 1994

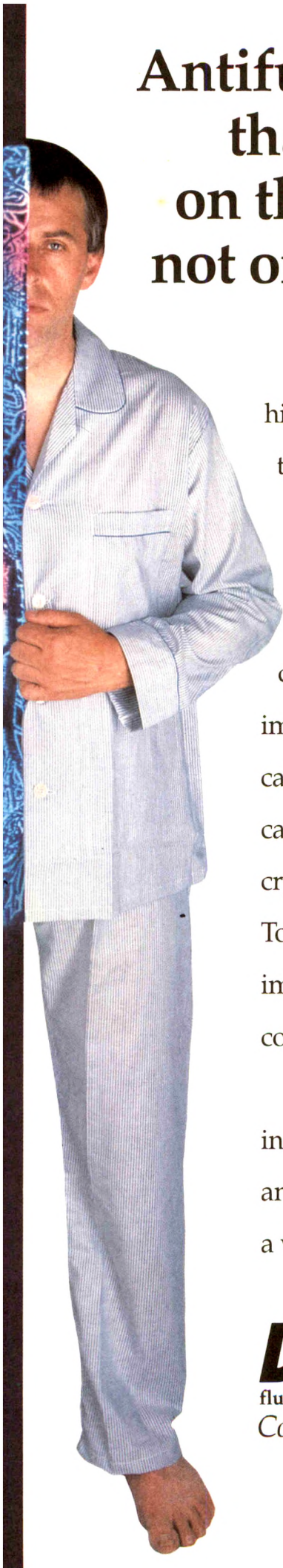


The Association of Anaesthetists of Great Britain and Ireland  
*Patron: HRH The Princess Margaret, Countess of Snowdon*



Published for the Association by  
Academic Press Grune & Stratton  
London San Diego New York  
Sydney Tokyo Toronto

ISSN 0003-2409



# Antifungal therapy that's severe on the infection, not on the patient.

Diflucan Oral/I.V. is a highly effective, well tolerated therapy with a convenient, once a day dosage and administration.

Overall clinical responses in deep-seated fungal infections are impressive: 87% in systemic candidiasis<sup>1</sup>, 96% in mucosal candidiasis<sup>2</sup> and 62% in cryptococcal meningitis.<sup>3</sup> Tolerability is similarly impressive with adverse effects comparable to placebo.<sup>4</sup>

Diflucan Oral/I.V. Treating invasive fungal infection with an iron fist and patients with a velvet glove.

**Diflucan** ORAL/I.V.  
fluconazole \*Trade Mark

*Counters invasive fungal infection*

Acc 1d 7  
13

# Anaesthesia

Journal of the Association of Anaesthetists of Great Britain and Ireland

---

Editor M. MORGAN

Assistant Editors R. GREENBAUM, A. R. AITKENHEAD, D. E. F. NEWTON, R. A. MASON, M. HARMER

*Editorial Board*

Chairman The President of the Association P. J. F. BASKETT

Members A.-M. ROLLIN, C. A. B. MCLAREN and a representative of the Junior Anaesthetists' Group

*Ex officio*

The Honorary Treasurer of the Association W. R. MACRAE

The Honorary Secretary of the Association J. E. CHARLTON

The Immediate Past Honorary Secretary of the Association W. L. M. BAIRD

---

DIANE WINTERBURN, JANET SPRINGER, Reference librarians

---

*Address for literary contributions* Dr M. Morgan, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, UK [telephone 081-740 3290].

Neither the Editor nor the Association of Anaesthetists of Great Britain and Ireland is in any way responsible for the statements made or the views expressed by contributors.

Investigations performed on man must conform to appropriate ethical standards including voluntary, informed consent and acceptance by an ethics committee.

A written statement of this acceptance may be requested by the Editor.

---

ANAESTHESIA: ISSN 0003-2409, Volume 46 1991, published monthly by Academic Press at 24-28 Oval Road, London NW1 7DX, UK, for the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA. All **advertising** enquiries should be addressed to the Advertising Department, Anaesthesia, Harcourt Brace Jovanovich, 2nd Floor, 24-28 Oval Road, London NW1 7DX (Tel: 071-267 4466; Telex: 25775 ACPRES G; Fax: 071-482 2293). Annual subscription price including postage: £132 UK and US \$244 overseas. Personal subscription rate including postage: \$99. Subscription orders should be sent to Academic Press Limited, High Street, Fooks Cray, Sidcup, Kent DA14 5HP (Tel: 081-300 3322). Send notices of changes of address to the publisher at least 6-8 weeks in advance, including both old and new address.

Second class postage rate paid at Jamaica, NY 11431, USA.

Air freight and mailing in the USA by Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

USA POSTMASTERS: send change of addresses to ANAESTHESIA, c/o Publications Expediting Inc., 200 Meacham Avenue, Elmont, NY 11003, USA.

Printed in UK on acid-free paper.

Anaesthesia is now available on 16 mm microfilm and 105 mm microfiche from Serials Acquisitions Department, University Microfilms Inc, 300 North Zeeb Road, Ann Arbor, Michigan 48106, USA.

© 1991 The Association of Anaesthetists of Great Britain and Ireland. The appearance of the code at the bottom of the first page of a paper in this journal indicates the copyright owner's consent that copies of the paper may be made for personal or internal use, or for the personal or internal use of specific clients in the USA. This consent is given on the condition, within the USA, that the copier pay the stated per-copy fee through the Copyright Clearance Center Inc., 27 Congress Street, Salem, MA 01970, USA for copying beyond that permitted by Sections 107 or 108 of the U.S. Copyright Law. This consent does not extend to other kinds of copying, such as copying for general distribution, for advertising or promotional purposes, for creating new collective works, for resale or for copying or distributing copies outside the USA.

Origination by BPCC Whitefriars Ltd, Tunbridge Wells, and printed and bound in Great Britain by BPCC Wheatons Ltd, Exeter.



# Baillière's International Practice and Research CLINICAL ANAESTHESIOLOGY

## Editorial Board

A.R. Aitkenhead (U.K.), M.J. Cousins (Australia), J.F. Crul (The Netherlands), J.G. Jones (U.K.),  
J.F. Nunn (U.K.), L. Strunin (Canada), D. Thomson (Switzerland), H. Van Aken (Belgium)

*Baillière's Clinical Anaesthesiology* is the essential reference for the busy clinician. Each issue is devoted to a separate topic and provides comprehensive coverage from experts in the field. Containing up-to-the-moment reviews, this international reference provides an invaluable source of current clinical opinion, procedures and techniques in anaesthesiology, supported by relevant research data.

## THE 1991 ISSUES

### No. 1 INFECTION AND THE ANAESTHETIST

Guest Editors:  
C.P. Stoutenbeck (Holland)  
& H.K.F. Van Saene (UK)

### No. 2 INTRAVENOUS ANAESTHESIOLOGY - WHAT IS NEW?

Guest Editors: J.W. Dundee (N.Ireland)  
& J.W. Sear (UK)

### No. 3 KINETICS OF ANAESTHETIC DRUGS IN CLINICAL ANAESTHESIOLOGY

Guest Editor: P.F. White (USA)

## PREVIOUS ISSUE TITLES TO COMPLETE YOUR REFERENCE

### Volume 4, 1990

No. 1  
AUTOMATED ANAESTHETIC RECORDS  
Guest Editor: G. Kenny (U.K.)

No. 2  
INTENSIVE CARE:  
DEVELOPMENTS AND CONTROVERSIES  
Guest Editor: G. Dobb (Australia)

No. 3  
ANAESTHESIA FOR DAY CARE SURGERY  
Guest Editor: T.E.J. Healy (U.K.)

### Volume 3, 1989

No. 1  
ANAESTHESIA FOR THE COMPROMISED HEART  
Guest Editor: P. Foex (U.K.)

No. 2  
METABOLIC RESPONSE TO SURGERY  
Guest Editor: D.R. Bevan (Canada)

No. 3  
DEPTH OF ANAESTHESIA  
Guest Editor: J.G. Jones (U.K.)

## ACKNOWLEDGED BY THE MEDICAL PRESS...

"A plethora of clinically applicable facts and therapy"..."An excellent source of reference for all clinicians"..."Up to date"..."Well written"..."Practical"..."Well produced and illustrated"..."Readable"..."Well balanced"..."Excellent source of reference"..."Concise"..."Comprehensive references"..."Excellent value"..."Cost effective"...

Publication: Volume 5 1991 (3 issues)

Subscription Rate: £41.00 (U.K. and Eire)/£47.00 (All other areas). Single Issues: £27.50 (All areas)



Published by Baillière Tindall  
Harcourt Brace Jovanovich, Publishers



## ORDER FORM: Baillière's Clinical Anaesthesiology

Please return to: Journals Marketing Dept., Harcourt Brace Jovanovich Ltd.,  
24-28 Oval Road, London NW1 7DX, U.K. Tel: 071-267-4466

ISSN: 0950-3501

Publication: Volume 5, 1991 (3 issues)

Prepayment is required

☐ Please enter my subscription for Volume 5, 1991

Back volumes and issues are available  
at current year prices.

☐ Please send me \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

### PRICES:

Annual Subscription: £41.00 (U.K. and Eire)/£47.00 (All other areas).

Single Issues: £27.50 (All areas)

☐ I enclose a cheque for £.....

(made payable to Harcourt Brace Jovanovich Ltd.)

☐ I wish to pay by credit card Amount £.....

Diners Club / American Express / Visa (Barclaycard) /

MasterCard (please delete as appropriate)

Card No. \_\_\_\_\_

MasterCard users should add the numbers appearing above their name

Expiry Date \_\_\_\_\_

Signature \_\_\_\_\_

(Your signature is essential, especially when ordering by credit card)

Name \_\_\_\_\_

Address \_\_\_\_\_

\_\_\_\_\_

\_\_\_\_\_

Date \_\_\_\_\_ JMD/0291/A789



*From Needle up  
to Finger Tip...*



*... Always the Real Feeling!*

# PERIFIX®

Epidural Sets with  
Loss Of Resistance Device



FOR FURTHER DETAILS ON PERIFIX® WITH LORD COMPLETE AND RETURN THIS COUPON

Name \_\_\_\_\_  
 Title \_\_\_\_\_  
 Hospital \_\_\_\_\_  
 Department \_\_\_\_\_  
 City \_\_\_\_\_

For UK inquiries, please contact:

## B. Braun Medical Ltd.

Braun House, 13/14 Farmbrough Close  
 Aylesbury Vale Industrial Park, Stocklake  
 Aylesbury, Bucks. HP20 1DQ  
 Phone: (0296) 39 39 00  
 Fax: (0296) 43 57 14 · Telex: 837086 bbmuk g

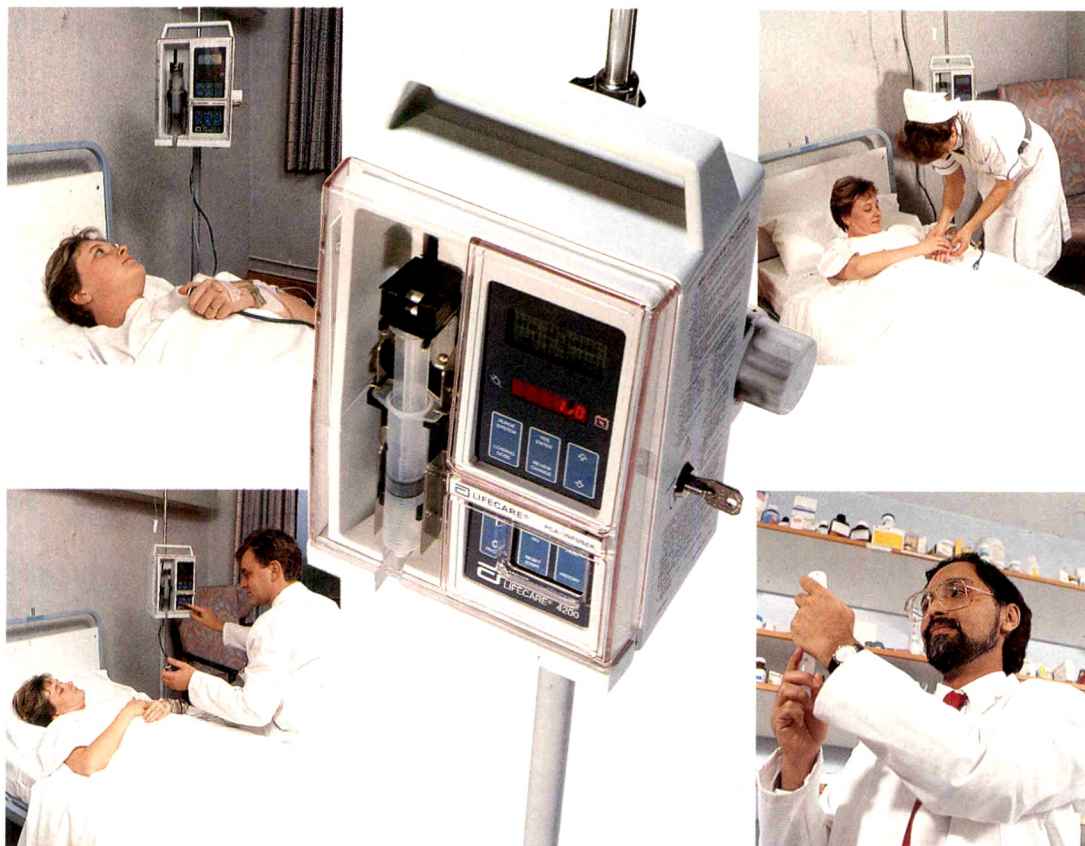
For international inquiries:

## B. Braun Melsungen AG

Medical Products Division  
 P.O.B. 110 + 120, 3508 Melsungen/Germany  
 Telephone (05661) 71-0



# ABBOTT PATIENT CONTROLLED ANALGESIA



## Allowing the patient membership of the pain management team...

- + Allows the patient to control their pain.
- + Allows the nurse time to manage the patient.
- + Allows the doctor to establish the dose.
- + Allows the pharmacist to monitor the drug.

If you would like to offer extra membership to your pain management team, please ask for more information from:

**ABBOTT**

**ABBOTT LABORATORIES LIMITED**  
Queenborough, Kent ME11 5EL  
Tel: (0795) 580099 Fax: (0795) 580404

**PCA+**



## Editorial

### The pill, HRT and postoperative thromboembolism: cause for concern?

During the last 20 years, clinical and laboratory evidence has accumulated which attributes administration of the synthetic oestrogen, ethinyl oestradiol, in the combined oestrogen/progestogen contraceptive pill to an increase in risk of overt, postoperative venous thromboembolism. This association has been extensively reviewed but with opposing conclusions. The 1985 editorial which appeared in the *British Medical Journal* recommended that oestrogen-containing pills be discontinued 4 weeks before major, elective surgery or any surgery to the legs, and that adequate, alternative contraceptive arrangements be made.<sup>1</sup> These recommendations, which do not apply to the progestogen-only 'mini' pill nor to minor procedures where the duration of surgery is short and early ambulation is anticipated, form the basis of current clinical practice and were incorporated into the *British National Formulary*. However, 3 years later the *British Medical Journal* published a further editorial which concluded that the combined pill should not be withheld from young women requiring abdominal surgery.<sup>2</sup> This conclusion, in turn, was criticised.<sup>3</sup>

The basis for this disagreement stems from markedly different interpretations of the relevant literature. The six epidemiological and clinical studies which form part of the basis of this debate were recently analysed by Hutchinson<sup>4</sup> and we recommend this paper to the interested reader. He reviewed their flaws which can be summarised thus. The two case-control studies, which reported that the relative risk of postoperative venous thromboembolism in pill users is increased at least two-fold after major surgery, were open to diagnostic-suspicion bias and patient-recall bias. Both relied on a clinical diagnosis which can be unreliable. The three prospective studies which employed <sup>125</sup>I-fibrinogen leg scanning for the diagnosis of deep venous thrombosis (DVT) failed to match the patient and control groups adequately for background factors likely to influence the risk of the disease (such as the weight of the patient and the duration of surgery and postoperative bed rest). The extent to which this omission confounded the results cannot be known, but one study reported that 19% of pill users developed a DVT (zero in controls), whereas another reported one DVT in 122 controls but none in the pill users. In the latter study, factors likely to increase the risk of postoperative DVT were concentrated in the control group. However, no explanation has been offered for the differences in the incidence of DVT in the pill users between these studies.

The sixth study of a long-term cohort provided such scant background details that Hutchinson<sup>4</sup> concluded that the information was impossible to interpret; however, other authors<sup>3</sup> have supported this work.

If the epidemiological and clinical data are open to more than one interpretation, is it biologically plausible that synthetic oestrogens increase the risk of venous thromboembolism? This results from blood coagulation and fibrin deposition in the presence of venous stasis.

Administration of synthetic oestrogens increases the biological activity of various procoagulant factors such as II, VII, X, XII and fibrinogen. However, the activities of the two most important coagulation inhibitors, antithrombin III and protein C, are not correspondingly increased. Indeed, antithrombin III activity is decreased and protein C appears unchanged. Whilst the extent to which these coagulation changes are opposed by alterations in the fibrinolytic system is unknown, it is well recognised that decreased antithrombin III or protein C activity is associated with thromboembolism.

Is it valid to extrapolate the coagulation changes induced in pill users by ethinyl oestradiol to postmenopausal women taking hormone replacement therapy (HRT) which is based on a 'natural' oestrogen? The numbers of women taking HRT has doubled during the last 2-3 years and the management problem that they pose will be encountered more frequently. Although no data are available on the risk of postoperative venous thromboembolism in HRT users, the incidence of spontaneous disease does not appear to be increased.<sup>5</sup> Because of the lack of epidemiological data, laboratory findings have assumed greater importance.

Factor VII, fibrinogen and antithrombin III levels rise when women go through menopause, and are higher than in age-matched premenopausal women.<sup>6</sup> If oestrogen status predicts antithrombin III activity then because administration of the pill results in a reduction in activity, menopause would be expected to result in an increase. However, this suggestion cannot explain the changes in factor VII and fibrinogen because menopause and use of the combined pill both result in an increase in these factors. This paradox emphasises that not only are the relationships between oestrogens and fibrinolytic/coagulation mechanisms extremely complex, but also that administration of the combined pill does not produce changes which are the reverse of those observed with loss of endogenous oestradiol at menopause. Much the most likely explanation for this dichotomy resides in the nature of the oestrogen in the pill, and the route of administration.

For reasons beyond the scope of this editorial, substitution of an ethinyl group at the C-17 position of the oestradiol molecule greatly enhances the potency of the oestrogen, ethinyl oestradiol. On a weight-for-weight basis, ethinyl oestradiol is approximately 600 times more potent than the natural oestrogen, oestrone sulphate, with respect to induction of sex hormone binding globulin.<sup>7</sup> The latter is produced within hepatic tissue: so are many fibrinolytic/coagulation factors. Furthermore, oral administration of oestrogen results in the absorbed steroid being delivered as a bolus to the liver,<sup>8</sup> whereas nonoral, oestrogen delivery systems (patches and implants) do not. The effect of natural oestrogens in HRT on triglycerides is a good example of how the route of administration determines the biological response: oral HRT increases plasma triglycerides, whereas nonoral routes result in a reduction.<sup>9</sup> Cross-

over studies comparing oral ethinyl oestradiol 10 µg/day (one third of the dose used in the pill), and oral oestradiol valerate (a natural oestrogen) 2 mg/day in postmenopausal women reported that the former causes significant increases in factors VII and VIII and a significant reduction in antithrombin III. The only effect of the natural oestrogen was to reduce antithrombin III.<sup>10</sup> This effect of oral oestradiol valerate on antithrombin III was confirmed by another study which also reported that percutaneous (nonoral) administration of oestradiol from a skin cream was without effect.<sup>11</sup>

How then should the user of an oestrogen containing pill awaiting major surgery or any surgery to the legs be advised? Although the current evidence has been interpreted in two ways, the recommendation in the *British National Formulary*, especially in the present increasingly litigious environment, bodes ill for patient and doctor alike (but not for the lawyer) should such pill usage be continued and a postoperative venous thromboembolism occur. An interval of at least 4 weeks seems necessary to allow for the potentially thrombotic haemostatic changes that occur during combined pill administration to be corrected.<sup>12</sup> Alternative methods of contraception have to be offered and the progestogen-only 'mini' pill or depo-progestogen injection, which do not appear to increase the risk of postoperative venous thromboembolism, seem suitable if barrier methods are unacceptable. Other strategies must be considered when emergency surgery is required and these have recently been reviewed.<sup>13</sup> However, it remains unclear whether the potential advantages of subcutaneous heparin outweigh the disadvantages.

The postmenopausal woman discontinuing HRT 4–6 weeks before surgery will not become pregnant but she may experience a recurrence of depressed mood, frequent daytime flushes and nocturnal sweats and other unpleasant menopausal symptoms. Illogically, the *British National Formulary* recommendation concerning discontinuation of the combined pill also applies to HRT, but on what evidence is this based? Presumably, it is because they both contain oestrogen. However, oral HRT appears to cause much smaller changes in fibrinolytic/coagulation mechanisms than the pill. Nonoral HRT delivers oestradiol in a manner similar to the ovary into the systemic circulation and achieves plasma values similar to those observed in a woman of reproductive age during the normal menstrual cycle. If physiological replacement of oestradiol with nonoral HRT is considered to be potentially so damaging, then all premenopausal women should be rendered medically menopausal by an LHRH supra-agonist for 4–6 weeks prior to relevant surgery.

Department of Anaesthetics,  
The Middlesex Hospital,  
London W1N 8AA  
Academic Department of  
Obstetrics and Gynaecology,  
King's College School of  
Medicine and Dentistry,  
London SE5 8RX

E. M. WHITEHEAD

M. I. WHITEHEAD

## References

1. GUILLEBAUD J. Surgery and the pill. *British Medical Journal* 1985; **291**: 498–9.
2. SUE-LING H, HUGHES LE. Should the pill be stopped preoperatively? *British Medical Journal* 1988; **296**: 447–8.
3. GUILLEBAUD J. Should the pill be stopped preoperatively? *British Medical Journal* 1988; **296**: 786–7.
4. HUTCHINSON GL. Oral contraception and post-operative thromboembolism: an epidemiological review. *Scottish Medical Journal* 1989; **34**: 457–9.
5. BOSTON COLLABORATIVE DRUG SURVEILLANCE PROGRAM. Surgically confirmed gallbladder disease, venous thromboembolism and breast tumors in relation to postmenopausal estrogen therapy. *New England Journal of Medicine* 1974; **290**: 15–9.
6. MEADE TW, DYER S, HOWARTH DJ, IMESON JD, STIRLING Y. Anti-thrombin III and procoagulant activity: sex differences and effects of the menopause. *British Journal of Haematology* 1990; **74**: 77–81.
7. MASHCHAK CA, LOBO RA, DOZONO-TAKANO R, EGGENA P, NAKAMURA RM, BRENNER PF, MISHELL DR. Comparison of pharmacodynamic properties of various estrogen formulations. *American Journal of Obstetrics and Gynecology* 1982; **144**: 511–8.
8. CAMPBELL S, WHITEHEAD MI. Potency and hepato-cellular effects of oestrogens after oral, percutaneous and subcutaneous administration. In van Keep PA, Utian WH, Vermeulen A, eds *The controversial climacteric*. Lancaster: MTP Press, 1982: 103–26.
9. BASDEVANT A, DE LIGNIERES B, GUY-GRAND B. Differential lipemic and hormonal responses to oral and parenteral 17β-estradiol in postmenopausal women. *American Journal of Obstetrics and Gynecology* 1983; **147**: 77–81.
10. LINDBERG UB, CRONA N, STIGENDAL L, TEGER-NILSSON AC, SILFVERSTOLPE G. A comparison between effects of estradiol valerate and low dose ethinyl estradiol on haemostasis parameters. *Thrombosis and Haemostasis* 1989; **61**: 65–9.
11. DE LIGNIERES B, BASDEVANT A, THOMAS G, THALABARD J-C, MERCIER-BODARD C, CONARD J, GUYENE T-T, MAIRON N, CORVOL P, GUY-GRAND B, MAUVAIS-JARVIS P, SITRUK-WARE R. Biological effects of estradiol-17β in postmenopausal women: oral versus percutaneous administration. *Journal of Clinical Endocrinology and Metabolism* 1986; **62**: 536–41.
12. ROBINSON GE, BURREN T, MACKIE IJ, BOUNDS W, WALSH K, FAINT R, GUILLEBAUD J, MACHIN SJ. Changes in haemostasis after stopping the combined contraceptive pill: implications for major surgery. *British Medical Journal* 1991; **302**: 269–71.
13. GOUCKE CR. Prophylaxis against venous thromboembolism. *Anaesthesia and Intensive Care* 1989; **17**: 458–65.

## Editorial notices

Manuscripts must be submitted in accordance with the internationally recognised *Uniform requirements for manuscripts submitted to biochemical journals* (*British Medical Journal* 1979; **1**: 432–5). Details will be found in the Notice to Contributors to *Anaesthesia* at the end of this issue.

## Cost of intensive therapy

### A description of methodology and initial results

S. RIDLEY, M. BIGGAM AND P. STONE

#### Summary

*A preliminary study was performed to calculate the cost of intensive therapy on an individual patient basis. The fixed (equipment, supporting services and land opportunity), semi-fixed (staff) and marginal (treatment) costs of 20 critically ill patients were calculated individually. The results show that there is wide variation in intensive therapy costs. The average daily cost for a spontaneously breathing patient was £399 (95% confidence intervals £388–£460) while that for a ventilated patient was £726 (£656–£795). The mean total cost per patient was £1980, but the cost per survivor increased by 16% (£347) because of four deaths on the intensive care unit. High total costs are associated with increased severity of illness and higher marginal (treatment) costs are associated with increased semi-fixed (staff) costs. The cost of intensive therapy was three to five times that for general ward care.*

#### Key words

*Intensive care; cost.*

The increased emphasis on efficient use of resources has focused attention on the relationship between the costs and benefits of health care programmes.<sup>1</sup> Simple estimates of treatment costs are the initial steps in cost-benefit analysis. A wide variety of illnesses is treated on general intensive therapy units (ITUs), and average costs derived from total expenditure and number of patient-days mask large cost differences between individual patients. In the USA, Chassin<sup>2</sup> reported that individual patient costs varied greatly; a small proportion of patients consumed a large share of total expenditure. Studies performed overseas have examined costs and outcome<sup>3,4</sup> but these results may not be strictly applicable to the UK because of differences in patient selection and quality of supporting facilities.

The aims of this preliminary study were to cost daily ITU treatment on an individual patient basis and to refine the method for use in a larger study.

#### Patients and methods

Twenty patients admitted consecutively to the ITU at the Western Infirmary, Glasgow, in March 1989 were studied prospectively. Details of each patient's age, diagnosis, duration and outcome of ITU treatment were recorded. The patient's severity of illness on admission was assessed using the Acute Physiology and Chronic Health Evaluation (APACHE) scoring system.<sup>5</sup>

The cost of intensive therapy may be divided into three main categories:

#### Fixed costs

The fixed costs are the capital costs, which include the purchase and maintenance of equipment and buildings and the supporting services such as portering and administration, and are incurred whether the ITU is occupied or not.

The land opportunity cost takes into account the value of the floor area of the ITU if it were to realise its full commercial value. An estimate, based on the vacant rental value, was provided by the Health Board.

Estimates of the daily costs of supporting services, administration and utilities for the Western Infirmary, Glasgow, are available from the Common Services Agency.<sup>6</sup>

An inventory with the date and purchase price of major items of equipment such as ventilators, infusion pumps and defibrillators is kept on the ITU. A 6% discount rate was used to account for the effect of inflation on the annual equivalent cost of equipment bought before 1989;<sup>7</sup> the contract maintenance fees for 1989 were added to the annual equivalent cost.

#### Semi-fixed

The semi-fixed costs are the staff costs. There are two main elements:

*Nursing staff.* The Intensive Care Society of Great Britain has recommended a dependency point classification of nursing support for intensive therapy.<sup>8</sup> The dependency points range from 2 for the most seriously ill patient to

S. Ridley, FFARCS, Senior Registrar, P. Stone,\* FFARCS, Registrar, Department of Anaesthetics, M. Biggam, SRN, Senior Nurse, Intensive Therapy Unit, Western Infirmary, Glasgow G11 6NT.

Correspondence should be addressed to S. Ridley please.

\*Present address: Senior Registrar, University Hospital of Wales, Heath Park, Cardiff, CF4 4XW.

Accepted 5 November 1990.

0.5 for a patient who needs little nursing care. The dependency points were allocated on a daily basis by a senior ITU nurse. A method for assigning nursing costs according to dependency point classification was recommended by Sheill (personal communication). The gross combined salaries of the nursing staff present on each of the study days were calculated from records of daily work rosters and pay scales (including overtime). The nursing staff cost per patient dependency point was calculated for each day. An estimate of the costs for nursing care for each patient was obtained by multiplying the cost per dependency point by the number of dependency points ascribed to that patient.

**Medical staff.** The medical staff were devoted exclusively to patients on ITU, where they performed a wide range of clinical duties. The difficulties in measuring medical time accurately led us to assume that medical staff input should parallel that of the nursing staff. Thus, the medical staff cost per dependency point was calculated in the same way as for the nursing staff.

Other staff, such as ward receptionist, were included in the supporting services in the fixed costs.

### Marginal costs

Marginal costs are the costs of the patient's treatment. On each day, the type of ventilatory support, the number and type of invasive lines, the surgical procedures carried out in ITU or theatres, the investigations performed, the drug doses and the fluids administered were recorded for each patient.

Estimates of treatment costs were obtained from various laboratory departments. The costs of biochemistry, virology and microbiology, which included estimates for disposable items, staff and equipment, were derived from in-house cost analyses performed over the same period. Estimates for haematological services were not available and so costs for these investigations were based on charges levied in the private sector. Costs for blood products are available from the Blood Transfusion Service.<sup>9</sup> Accurate hospital costs of radiological investigations were not available; consequently, the costs were based on Government-agreed fees for various classes of radiological investigation.<sup>10</sup> Prices for drugs, fluids and disposable equipment, such as ventilator tubing and intravenous lines, were available from the Pharmacy Department.

### Statistical methods

Analysis of variance was used to examine whether the costs varied significantly among patients.<sup>11</sup> If a significant patient effect was found, only one value from each patient was used in further statistical analysis. However, if no significant interpatient variability was present, the data were treated as independent. A *p* value of less than 0.05 was considered significant.

### Results

The median age of the patients was 52 years; the 10th and 90th centiles were 20 and 73 years respectively. The APACHE scores ranged from 0 to 28; the median, 10th and 90th centiles were 10, 0 and 27. The median duration of admission was 3 days (10th and 90th centiles = 1 and 13

days). The diagnosis was related primarily to the respiratory system in nine patients, the cardiovascular system in four patients, and the gastrointestinal system in two patients; five patients were admitted as a result of trauma (including one with a fractured mandible). Half of the patients were ventilated at some time during their admission.

The total daily fixed cost for each patient was £82 (Table 1). The daily equipment costs are derived from the annual equivalents of the cost of purchase, discounted to 1989 values and the maintenance costs discounted for 1989. Because the defibrillator and blood gas analyser are available to all patients, the daily capital costs of these machines are based on an average bed occupancy of five patients. During March 1989, there were 155 patient-days of treatment on the ITU and so apportioning the land opportunity cost equally, the cost per patient-day is £15.

Table 2 shows the daily dependency point assignment for each patient. It is important to note that patients not included in the study contributed to the total nursing dependency points at the beginning and end of the study period. Table 3 shows the nursing costs for each patient on each day. The combined gross nursing salaries for each day are shown at the top of the table. The nursing staff cost per dependency point and the patient's individual dependency point assignment (Table 2) were used to calculate each patient's nursing staff costs. The average cost per nurse per day is relatively constant (mean £60, 95% CI £52–62) despite the daily variations in the composition of the nursing staff with respect to seniority and rates of pay.

The medical cover for the ITU consists of five consultants, with a combined commitment of 23 sessions per week, one senior registrar, one registrar and two senior house officers. The gross combined salaries for the medical staff for each day is £441 (taking account of incremental pay scales, merit award and overtime payments) and this has been allocated in the same way as nursing costs (Table 4).

The marginal costs for supportive treatment and investigations were calculated daily and an example of the summary of one patient's daily treatment costs is given in Table 5. Table 6 gives the treatment costs for all 20 patients for each day.

The total daily ITU treatment cost is a sum of the fixed, semi-fixed and marginal costs (Table 7). Analysis of variance revealed that there was no significant patient effect for those patients who breathed spontaneously throughout their ITU admission (mean daily cost £399, 95% CI

Table 1. Fixed costs (per patient per day).

<b>Equipment</b>	
Monitoring (ECG, pressure monitoring)	£5.24
Ventilators (including humidifiers)	£5.93
Infusion devices	£0.63
Defibrillator, blood gas analyser	£3.35
<b>Services</b>	
Services (catering, portering, laundry)	£19.00
Administration (medical, nursing, general)	£15.14
Property (maintenance, cleaning, heating)	£18.00
<b>Land opportunity cost</b>	
£10* per ft <sup>2</sup> year for a 2702 ft <sup>2</sup> ITU	
155 patient-days in March 1989	£15.00
<b>Total</b>	<b>£82.29</b>

\*Greater Glasgow Health Board estimate of vacant rental value.

Table 2. Daily nursing dependency points for each patient.

	Day																																										
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26																	
	Other patients' total																																										
Patient	3.5	3	1.5	1.5	1.5	1.5	1.5	1	1	1	1	1	1	0.5	0.5	0.5	0.5																										
1	1	1	1	1	1	0.5	0.5																																				
2	1	0.5	0.5	0.5	0.5																																						
3		1	0.5	0.5	0.5																																						
4							1.5																																				
5							1.5	2																																			
6							1	1	1	0.5	0.5	0.5	0.5																														
7								1	1	0.5	0.5																																
8								1	0.5																																		
9									1.5	1	1	1	1	1	0.5	0.5																											
10										1.5																																	
11											1	0.5																															
12											0.5	0.5																															
13												1.5	1.5																														
14												1	0.5																														
15												1	0.5	0.5																													
16													1.5	1	1.5	1.5	1.5	1	1	1.5	1	1	1	0.5																			
17														1	0.5	0.5																											
18															1	0.5																											
19																1	0.5	0.5																									
20																	1	0.5																									
																		Other patients' total																									
																		2	2.5	3	1.5	2	5	2.5	7	5.5	5.5																
																		Total nursing dependency points																									
																		5.5	5.5	3.5	3.5	3.5	2	6	6	5	4.5	4.5	5	5	5.5	4	5	5.5	5	4	2.5	3.5	6	3.5	8	6.5	6

Table 3. Semi-fixed costs: daily nursing staff costs for each patient apportioned according to dependency point allocation.

	Day																									
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
Gross daily nursing salaries																										
	856	751	985	983	906	983	1042	1240	1054	1093	943	1178	976	1083	825	1056	943	964	1058	978	968	969	1025	1092	899	982
Total nursing dependency points																										
	5.5	5.5	3.5	3.5	3.5	2	6	6	5	4.5	4.5	5	5	5.5	4	5	5.5	5	4	2.5	3.5	6	3.5	8	6.5	6
Nursing staff costs per dependency point																										
Patient	156	137	281	281	259	492	174	207	211	243	210	236	195	197	206	211	171	189	265	391	277	162	293	137	138	164
1	156	137	281	281	259	246	87																			
2	156	69	141	141	130																					
3		137	141	141	130																					
4							261																			
5							261	414																		
6							174	207	211	122	105	118	98													
7								207	211	122	105															
8								207	106																	
9									317	243	210	236	195	197	103	106										
10										365																
11											210	118														
12											105	118														
13												354	293													
14													195	99												
15														197	103	106										
16														296	206	317	257	284	265	391	416	162	293	137	138	82
17														197	103	106										
18															206	106										
19																211	86	95								
20																	171	95								

(Costs approximated to the nearest whole pound, *italicised* figures signify mechanical ventilation on that day.)

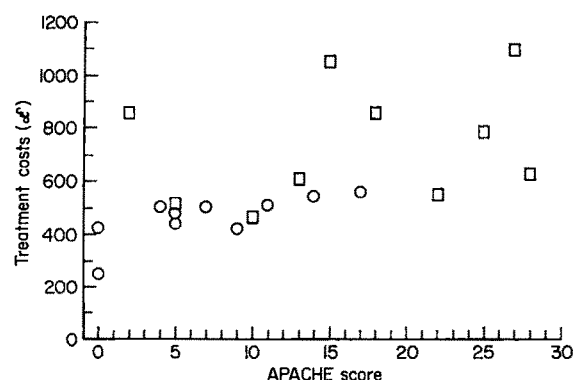


Fig. 1. Scatter diagram of the total intensive therapy costs (in pounds) varying with severity of illness on admission for all patients. (Squares represent ventilated patients; circles represent spontaneously breathing patients).

£338–460) and those who had been weaned from mechanical ventilation (mean daily cost £440, 95% CI £270–610). However, there was significant interpatient variability for the days when mechanical ventilation was required ( $p < 0.001$ ). The mean (95% CI) cost for each day of mechanical ventilation was £726 (£656–795).

The mean daily cost for survivors was £444 (£371–517) while that for nonsurvivors was £926 (£840–1012). The average total cost per patient was £1980 but this varied between a mean of £2028 for survivors and £1389 for nonsurvivors, a reflection of the shorter duration of stay of nonsurvivors. The average cost for each patient who left the ITU alive should increase by £347 on account of the cost incurred by the four patients who died.

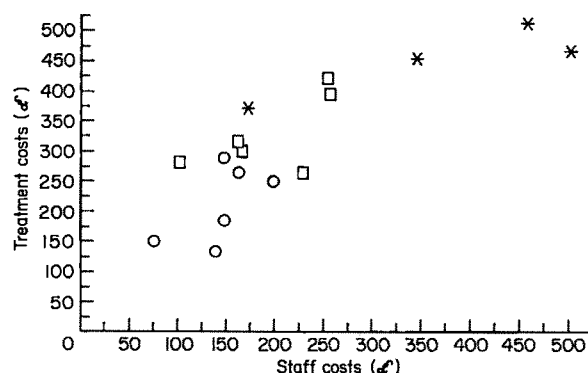


Fig. 2. A scatter diagram of the mean daily staff and treatment (marginal) costs (in pounds) for the 10 ventilated patients. (Asterisks represent four nonsurvivors. Six patients were weaned from respiratory support and for these six, squares represent days of mechanical ventilation while circles indicate spontaneous respiration).

Figure 1 is a scatter diagram of the first day's total costs for all patients in relation to the APACHE score. Regression analysis shows that total cost may be predicted from APACHE score (Cost = £433 + (14.3 × APACHE score); CI of coefficients, constant = 285–581, APACHE score = 4.2–21.2,  $p < 0.05$ ).

Figure 2 is a scatter diagram of the mean daily staff and treatment costs for the ventilated patients. For those patients who were weaned from mechanical ventilation, the mean staff and treatment costs for days with and without respiratory support are shown separately. Regression analysis revealed a significant association between staff and treatment costs (staff cost = £145 + (0.8 × treatment costs);

**Table 4.** Semi-fixed costs: daily medical staff costs for each patient apportioned according to dependency point allocation. Cost of medical staff per dependency point = £441/(total daily dependency points). Cost of medical staff for any particular patient = (cost of medical staff per dependency point) × (patients dependency point allocation).

Patient	Day																									
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
	Total daily dependency points																									
	5.5	5.5	3.5	3.5	3.5	2	6	6	5	4.5	4.5	5	5	5.5	4	5	5.5	5	4	2.5	3.5	6	3.5	8	6.5	6
Medical staff costs per dependency point																										
	80	80	126	126	126	221	74	74	88	98	98	88	88	80	110	88	80	88	110	176	126	74	126	55	68	74
1	80	80	126	126	126	111	37																			
2	80	40	63	63	63																					
3		80	63	63	63																					
4						111																				
5						111	148																			
6						74	74	88	49	49	44	44														
7							74	88	49	49																
8							74	44																		
9								132	98	98	88	88	80	55	44											
10									147																	
11										98	44															
12										49	44															
13											132	132														
14												88	40													
15													80	55	44											
16													120	110	132	120	132	110	176	189	74	126	55	68	37	
17													80	55	44											
18														110	44											
19															88	40	44									
20																80	44									

(Costs approximated to the nearest whole pound; italicised figures signify mechanical ventilation on that day).

Table 5. An example of the summary of one patient's daily treatment.

Day number and name:	Biochemistry	Microbiology	Haematology	Virology	Drug name or code	Drug cost	Doses	Total drug cost	Fluids
Ventilation	8	1	1		1	3	3	9	4
Arterial line	9	11			Diamorphine and	7.51	5	37.55	11
CVP lines	1				Bupivacaine	0.06	1	0.06	
Pulmonary arterial lines	2				Metoclopramide	1.84	4	7.36	
Peripheral lines	3				13				
Other lines	4								
N + CDW/M	5								
Monitoring total	30.41								
Totals	24.00	12.68	10.00	0				53.97	1.06
Surgery	Nil								
Radiology	C								
Renal support	Nil								
Investigations	46.68								
Drugs/fluids	55.03								
Total treatment	171.91								

Figures given as pounds.

Monitoring:

S/N = spontaneous respiration

A/C = arterial monitoring

P/IF = peripheral line for clear

fluids inserted

N = nasogastric tube

CDW/M = daily maintenance to under-

water chest drain.

C = chest X ray

Biochemistry

1 = Biochemical profile

3 = Glucose

4 = Serum osmolality

5 = Cortisol

8 = Emergency urea and electrolytes

9 = Emergency creatinine

Microbiology: 1 = Selective decontamination of digestive tract screen

11 = C-reactive protein

Haematology: 1 = Full blood count

Drug code: 1 = Cefotaxime 500 mg

13 = Antimicrobial agents for selective decontamination of digestive tract

Fluids:

4 = 500 ml glucose 5%

11 = 500 ml saline 0.9%



**Table 6.** Marginal costs: the individual costs for investigations and supportive treatment for each patient.

Patients	Day																									
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
1	195	198	145	110	172	113	159																			
2	122	172	141	75	138																					
3		124	182	123	82																					
4							173																			
5							645	362																		
6							278	180	262	138	101	110	130													
7								140	195	126	137															
8								102	76																	
9									328	188	150	209	203	397	149	91										
10										459																
11											170	49														
12											15	31														
13												290	405													
14													59	75												
15														145	123	96										
16														290	155	269	443	246	273	204	185	203	176	125	126	163
17														117	91	98										
18															147	151										
19																167	198	81								
20																	178	123								

(Costs approximated to the nearest whole pound; italicised figures signify mechanical ventilation on that day.)

**Table 7.** Total daily treatment costs for each patient.

Patients	Day																									
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26
1	513	497	634	599	639	552	365																			
2	440	363	427	361	413																					
3		423	468	409	357																					
4 (died)							627																			
5 (died)							1099	1006																		
6							608	543	643	391	337	354	354													
7								503	576	379	373															
8								465	308																	
9									859	611	540	615	568	756	389	323										
10 (died)										1053																
11											560	293														
12											251	275														
13 (died)												858	912													
14													424	296												
15														504	363	328										
16														788	553	800	902	744	730	853	872	521	677	399	414	364
17														476	331	330										
18															545	383										
19																548	406	302								
20																	511	344								

(Costs approximated to the nearest whole pound; italicised figures signify mechanical ventilation on that day).

CI of coefficients, constant = 70–220, treatment costs = 0.5–1.1,  $p < 0.01$ ). A similar analysis for the spontaneously breathing patients failed to reach statistical significance.

### Discussion

Comprehensive cost description of any health programme should consider three areas of cost: the organising and operating costs within the health care sector; the costs incurred by the patients and their families; and costs borne outside the health care sector, patients and families, such as employment-related health insurance.<sup>7</sup> The costs borne by the patient and families are undoubtedly important, but

the aim of this study was limited to estimate the cost of ITU treatment alone. This study did not calculate the remaining hospital costs or those incurred once the patient returned to the community. Intensive care is an important component of hospital costs; Bam and Miranda<sup>3</sup> reported that 52% of the total hospital costs were generated by ITU treatment although the time spent in ITU accounted for only 17.5% of the total admission time. Intensive therapy competes with other hospital based programmes, and estimates of ITU costs alone, as opposed to total costs including community care, may be worthwhile.

Only the major components of ITU treatment were considered. Stoddart<sup>12</sup> has pointed out that it is not only

difficult but also not worthwhile, in view of time and effort expended, to attempt to estimate all the minor costs associated with a particular programme. Therefore the estimates provided by the Common Services Agency were used.

Other studies of ITU treatment costs have adopted either a top-down approach<sup>13</sup> or graded the nursing care according to the nurse:patient ratio.<sup>14</sup> Top-down studies, which start with the total budget and work down to produce average costs per patient, are worthwhile when all patients conform to a similar pattern in respect of severity of illness or diagnosis. The use of fixed nurse:patient ratios may be influenced by staffing levels rather than patient requirement.

Calculations for semi-fixed costs based upon the combined gross salaries for all the staff were considered more valuable than basing estimates solely on the personnel who actually treated the patient. Other nurses on the ward contribute to the patients' care in terms of practical help, supervision and meal relief. Also while the aim was to cost each patient's treatment individually, an element of this cost includes a share of the costs of a fully staffed ITU. However, allocation of staff costs using a nursing dependency classification means that the daily treatment costs are influenced by the total number of dependency points. This may have introduced some bias in a small study such as this, but the effect of fluctuation in workload should decrease in larger studies.

The average daily cost of a staffed bed on a general ward at the Western Infirmary is £145,<sup>6</sup> suggesting that intensive therapy is very expensive (almost three times the general ward level for a spontaneously breathing patient and almost five times for a mechanically ventilated patient). These results agree with those of Sheill *et al.*, who reported the daily cost of intensive therapy in the UK as £500 per patient with a total average cost of £2000 per patient.<sup>15</sup> In the USA, intensive therapy has been reported to be 3.8 times more expensive than general ward care.<sup>16</sup>

The high cost of treatment for those patients who are less ill and did not require ventilatory support is an important finding because Ron and colleagues<sup>17</sup> have suggested that only a small proportion of such patients may actually benefit from intensive therapy. In a large study of 5000 patients, Knaus *et al.*<sup>18</sup> reported that 24% of patients were admitted to ITU for observation and monitoring but that only 10% received active 'intensive therapy' interventions. Therefore patients who are less ill may be more appropriately managed on a high dependency unit (HDU) where the staff costs are lower. The combination of HDUs and ITUs in the same hospital, with the ITU reserved for treatment of the most seriously ill, has been recommended recently.<sup>19</sup>

The results suggest that ITU expenditure depends upon severity of illness, level of intervention and ITU mortality. Significantly higher costs for nonsurvivors have been reported in the USA.<sup>20</sup> In this study, a 20% mortality rate increased the total cost for each survivor by 16% but a higher figure could be expected if patients die after a prolonged critical illness.

Future costing exercises need not be as complicated. There is minimal variation in the fixed costs and in the daily average staffing cost per nurse so that only the nurse dependency score and marginal costs need to be recorded. The data for marginal costs should be available for any

ITU which carries out a reasonable level of audit. Another advantage of such methodology is that it contains building blocks which can then be used to investigate the cost implications of any changes in ITU management.

Patients should not be denied intensive therapy on the basis of financial considerations, although these results, illustrating the high level of ITU expenditure, may act as a stimulus for the more widespread use of either careful case selection or accurate predictive models for ITU outcome. Several models have been described; most involve sequential scoring of either severity of illness or therapeutic interventions.<sup>21,22</sup> However such models are not used widely in the UK. The accurate prediction of eventual ITU mortality is important so that when the outlook is known to be hopeless, close relatives may be spared further distress, and the patient spared a prolonged death.

Future work directed at accurate prediction of outcome is required to ensure that resources are appropriately used. In addition, the financial implications of increased use of HDUs should be explored.

### References

1. KNAUS WA. Too sick and old for intensive care. *British Journal of Hospital Medicine* 1987; **37**: 381.
2. CHASSIN M. Costs and outcomes of medical intensive care. *Medical Care* 1982; **2**: 165-79.
3. BAM JL, MIRANDA DR. Outcome and costs of intensive care. *Intensive Care Medicine* 1985; **11**: 234-41.
4. LOES O, SMITH-ERICHSEN N, LIND B. Intensive care: Cost and benefit. *Acta Anaesthesiologica Scandinavica* 1987; **31** (Suppl. 84): 3-19.
5. KNAUS WA, DRAPER EA, WAGNER DP, ZIMMERMAN JE. APACHE II: A severity of disease classification system. *Critical Care Medicine* 1985; **13**: 818-29.
6. Common Services Agency. Scottish Health Service Costs. Edinburgh: Lothian Health Board, 1989.
7. DRUMMOND MF, STODDART GL, TORRANCE GW. *Methods for the economic evaluation of health care programmes*. Oxford: Oxford University Press, 1987.
8. Standards Sub-Committee. Intensive Care Society of Great Britain. *Standards for intensive care units*. London: BioMedica Ltd., 1983.
9. United Kingdom Blood Transfusion Services *Handbook of transfusion medicine*. London: Her Majesty's Stationery Office, 1989.
10. SIMMONS S. Fees update. *British Medical Association News Review* 1989; **15**: 29-30.
11. SNEDECOR GW, COCHRAN WG. *Statistical Methods*. 6th edn. Iowa University Press, 1967: 259-98.
12. STODDART GL, DRUMMOND MF. How to read clinical journals: VII. To understand an economic evaluation (part B). *Canadian Medical Association Journal* 1984; **130**: 1542-9.
13. BENDIXEN HH. The cost of intensive care. In: BUNKER JP, BARNES BA, MOSTELLER F, eds. *The costs, risks and benefits of surgery*. New York: Oxford University Press, 1977.
14. GIROTTI MJ, BROWN SJL. Reducing the costs of ICU admission in Canada without diagnosis-related or case-mix groupings. *Canadian Anaesthetists' Society Journal* 1986; **33**: 765-72.
15. SHEILL A, GRIFFITHS RD, MACMILLAN RR, ATHERTON ST. Pilot assessment of the cost-effectiveness of a British intensive care unit. *British Journal of Hospital Medicine* 1989; **42**: 144-5.
16. WAGNER DP, WINELAND TD, KNAUS WA. The hidden costs of treating severely ill patients. Charges and resource consumption in an intensive care unit. *Health Care Financial Review* 1983; **5**: 81-6.
17. RON A, ARONNE LJ, KALB PE, SANTINI D, CHARLSON ME. The therapeutic efficacy of critical care units. Identifying subgroups of patients who benefit. *Archives of Internal Medicine* 1989; **149**: 338-41.
18. KNAUS WA, DRAPER EA, WAGNER DP, ZIMMERMAN JE, BIRNBAUM ML, CULLEN DJ, KOHLES MK, SHIN B, SYNDER JV.

- Evaluating outcome from intensive care; a preliminary multihospital comparison. *Critical Care Medicine* 1982; **10**: 491-6.
19. Intensive Care Society of Great Britain. *The intensive care service in the UK*. London: Intensive Care Society, 1990.
  20. DETSKY AS, STRICKER SC, MULLEY AG, THIBAUT GE. Prognosis, survival and the expenditure of hospital resources for patients in an intensive-care unit. *New England Journal of Medicine* 1981; **305**: 667-72.
  21. CHANG RWS, JACOBS S, LEE B, PACE N. Predicting deaths among intensive care unit patients. *Critical Care Medicine* 1988; **16**: 34-42.
  22. JORDON DA, MILLER CF, KUBOS KL, ROGERS MC. Evaluation of sepsis in critically ill surgical population. *Critical Care Medicine* 1987; **15**: 897-904.

## Immunological disturbances in anaesthetic personnel chronically exposed to high occupational concentrations of nitrous oxide and halothane

M. PERIĆ, Z. VRANEŠ AND M. MARUŠIĆ

### Summary

*Immunological changes in anaesthetic personnel exposed to occupational concentrations of halothane and nitrous oxide 10–60 times greater than the advised maximum were studied during routine work and after 3–4 weeks holiday. Red cell count, haemoglobin concentration and haematocrit decreased during exposure although not significantly, in comparison with a control group, but all had increased significantly after the holidays. Other changes were altered neutrophils and lymphocyte counts. Basophils disappeared from the blood during the exposure. Monocytes were not affected during the exposure, but increased after its cessation. Percentages of CD2 and CD4 lymphocytes increased significantly, but numbers of cells in T lymphocyte subpopulations (total, helper and cytotoxic/suppressor lymphocytes) were not significantly altered. B lymphocytes were most strongly affected: they decreased during working periods and did not recover after holidays. Natural killer (NK) cells, on the other hand, decreased significantly during exposure, but fully recovered during holidays. After stimulation with mitogens, phytohaemagglutinin, concanavalin A, and pokeweed, lymphocytes from exposed personnel incorporated significantly more <sup>3</sup>H-thymidine than those from control subjects, but stimulation indices did not differ. The natural killer-cell activity, serum Ig concentrations and phagocytosis by granulocytes were not altered.*

### Key words

*Immune response.*

*Anaesthetic gases; trace concentrations.*

*Anaesthetics volatile; trace concentrations.*

Occupational exposure of anaesthetic personnel to anaesthetic gases has been widely investigated,<sup>1–13</sup> but information on how the immune system is affected is limited.<sup>14–16</sup> Nitrous oxide<sup>17</sup> and halothane<sup>18</sup> decreased the number of leukocytes in the peripheral blood of rodents, but allogenic skin transplant rejection was not affected.<sup>18</sup> Halothane and nitrous oxide in mice caused partial inhibition of cell-mediated cytotoxicity;<sup>19</sup> halothane decreased the responsiveness of human lymphocytes to phytohaemagglutinin.<sup>20</sup> Halothane also impaired lymphocytotoxicity against allogenic tumour cells of the same histological type in children.<sup>21</sup> Nunn's group reported that neutrophil migration, chemotaxis, phagocytosis, degranulation and enhanced non-mitochondrial respiration associated with phagocytosis were unaffected after *in vitro* exposure to halothane,<sup>22</sup> but nitrous oxide decreased motility of human neutrophils.<sup>23</sup> A similar negative finding for halothane was

reported by others,<sup>24,25</sup> but nitrous oxide was shown to increase polymorphonuclear leukocyte chemotaxis<sup>26</sup> and even counteract the adverse effect of halothane.<sup>27</sup> Some authors hold that *in vivo* inhibition of phagocytosis during anaesthesia results from factors such as stress or altered blood flow and not from the effects of anaesthetics.<sup>28</sup>

This present study investigates the effects of nitrous oxide and halothane on the immunological system of anaesthetic personnel. The staff had been complaining of weakness and recurrent infections and decreased peripheral blood leukocyte counts had often been found on routine control testing. We therefore checked the concentrations of halothane and nitrous oxide in operating theatres and determined some immunohaematological variables in the personnel at the peak of the working season and after the summer holidays. It turned out that the operating theatres were improperly ventilated, with increased concentrations

Immunology Division, Department of Clinical Laboratory Diagnostics, Zagreb University School of Medicine and Clinical Centre Zagreb, and Department of Anaesthesiology and Intensive Therapy of "Dr. Mladen Stojanovic" Clinical Hospital, Zagreb, Croatia, Yugoslavia.

Correspondence to Prof. dr. Matko Marusic, Immunology Division, Department of Clinical Laboratory Diagnostic, Clinical Centre Zagreb-Rebro, Kispaticeva 12, 41000 Zagreb, Croatia, Yugoslavia.

Accepted 24 September 1990.

of nitrous oxide and halothane in the working atmosphere. To our knowledge, this is the first comprehensive study of *in vivo* immunological effects of increased concentrations of nitrous oxide and halothane in man.

### Materials and methods

#### *Tested individuals*

Twenty-one staff members working at the Department of Anaesthesiology and Intensive Therapy (16 women and five men) who had been working at this institution for 8–20 years were tested. After the summer holidays, tests were conducted on only 17 members of staff (13 women and four men); four subjects were excluded, two for acute infection and two for other illnesses. The mean age in the group tested before and after the holidays was 40.7(SD 5.9) and 39(SD 6.3) years, respectively. To avoid the influence of X rays on the immune system we chose personnel who did not work in an X ray area.

We could find no group who worked under the same conditions as the tested individuals, but who were not exposed to waste gases. So we tested 35 healthy subjects from the same socio-economic class who work in offices. We tested 15 of them after the holidays (not shown). There were no significant differences in any of the variables and we used their results before the holidays as the control. To avoid any influence of duty or especially sleepless nights which might cause acute stress,<sup>29</sup> and influence the results, blood samples were taken on Monday and Thursday mornings after a free weekend and a peaceful night.

Anaesthetic personnel and subjects from the control group both spent their holidays at the seaside.

#### *Laboratory techniques*

Red and white cell counts, and haemoglobin concentration were determined using a Coulter S-plus device (Coulter, Hialeh, FL, USA). Lymphocyte subpopulations were determined using Coulter monoclonal antibodies and indirect immunofluorescence of peripheral blood. Mononuclear cells were separated from red cells and polymorphonuclear granulocytes by centrifugation on a ficoll (Ficoll-Paque, Pharmacia, Uppsala, Sweden) layer. Percentages of fluorescent cells were determined in the lymphocyte gate of an EPICS-C flow cytometer (Coulter). The natural killer (NK) cell activity was determined in a 4-hour <sup>51</sup>Cr-release assay from labelled K562 tumour cells.<sup>30</sup> Proliferation of lymphocytes after a 3-day cultivation with the mitogens phytohaemagglutinin (PHA), concavalin A (Con A) and pokeweed mitogen (PWM) was estimated by measuring the <sup>3</sup>H-thymidine incorporation in 0.2 ml-volume triplicate cultures of  $2 \times 10^5$  cells in 96-well culture plates (Nunc). Serum IgA, IgG, and IgM concentrations were determined by immunoelectrodifusion and IgD with radial diffusion (Behring-Partigen IgD). Phagocytosis by granulocytes was estimated by determination of an average number of yeast cells ingested per granulocyte after a 2-hour cocultivation.<sup>31</sup>

#### *Conditions in operating theatres*

None of the operating theatres have waste gas scavenging systems. (Two operating blocks have been built recently,

with modern scavenging equipment and venting of waste gases outside the building; this study, however, was carried out before they were completed.)

The concentrations of anaesthetic gases and vapours were determined in four, most frequently used operating theatres, two ENT and two surgical. One of the ENT theatres is regularly used for tonsillectomy in children and halothane, nitrous oxide and oxygen are used for anaesthesia. This theatre is equipped with a Draeger-Tiberius anaesthetic machine and halothane vaporizer, Vapor 19.3 Draeger (Draegerwerk A.G., Lübeck, Germany). The second ENT operating theatre is used for other ENT operations and is equipped with a new Draeger Sulla 808V anaesthetic machine with a halothane vaporizer Vapor, 19.3. The two operating theatres at the surgical clinic had Draeger Narcosespiromat 650 and Vapor-Halothan vaporizers manufactured in 1972. None of the theatres was equipped with a central gas supply system; continuous air-monitoring programmes did not exist in this hospital.

#### *Determination of occupational gas and vapour concentrations*

Infrared (IR) gas spectrometry was performed using a Miran 1A Wilks device (Foxboro, USA). The instrument monitors gas concentrations continuously for several hours, in various spots in the room, with a complete quantitative analysis every 3–5 seconds. The air flows continuously through a cell with a volume of 5.64 litres; the wavelength of the IR-beam is 4.41  $\mu$ m, with a path length of 20.25 m. The sensitivity of the instrument is 0.1 mg/m<sup>3</sup> and 0.05 ppm, respectively. In this study a 0.75 m path length of the IR-beam was used because of high concentrations of halothane and nitrous oxide in the atmosphere.

#### *Statistics*

Statistical analysis was performed on an IBM PC using a Statgraphics 1.2 programme. An unpaired Student's *t*-test was used to test differences between the control and experimental groups, before and after the holidays; a paired two-tailed Student's *t*-test was used to compare findings in the anaesthetic personnel obtained before and after vacations. Normality of data distribution was always tested by a Kolmogorov-Smirnov test; when the distribution was not normal (nonsegmented neutrophils, eosinophils, basophils, IgD), a nonparametric Wilcoxon's test was used to compare the paired, and a Mann-Whitney-Wilcoxon's test to compare the unpaired groups of data.

### Results

Immunohaematological variables in the anaesthetic personnel were determined during April and May 1988 (before the summer holidays) and after 3–4 weeks of holiday in July and August; the results that differed significantly from the controls in the first test were rechecked on the first working day after the holidays. Halothane and nitrous oxide concentrations were determined in April. As shown in Table 1, halothane time-weighted concentrations in the theatre in which it is used most frequently and at high concentrations (ENT-1, first line in Table 1), was 8–9

**Table 1.** Concentrations of halothane and nitrous oxide in the working area of anaesthetic personnel in 'Dr Mladen Stojanovic' Clinical Hospital in Zagreb.

Location	Gas and vapour concentrations*						
	Halothane (mg/m)			Nitrous oxide (ppm)			RH†(°C)
	Minimum	Maximum	TWA	Minimum	Maximum	TWA‡	
ENT 1§	264	2244	350	140	10000	1200-1500	26 50
ENT 2	—	—	—	115	453	350-400	23 49
Surgery 1¶	40	80	50	1250	2720	1600	24 44
Surgery 1**	—	—	—	400	1100	650	— —
Surgery 2††	10	40	30	900	1730	1400	24 45
Surgery 2§§	—	—	—	800	1070	900	—
Surgery 3	10	10	10	115	400	320	24 43
Surgery 4¶¶	—	< 10	—	80	100	85	22 44

\* In dental operating theatres the acceptable standard for halothane is 40 mg/m<sup>32</sup> and for nitrous oxide 50 ppm, whereas in other operating theatres nitrous oxide is acceptable at 25 ppm.<sup>33</sup>

† Relative humidity in the operating theatres.

‡ Time weighted average.

§ Operating theatre used for tonsillectomy in children where anaesthesia is performed with halothane at concentrations of 1.5-2.0 vol. %, and N<sub>2</sub>O and O<sub>2</sub> mixture at a 1:1 ratio. The data were obtained by measurements during three consecutive tonsillectomies.

|| Operating theatre for ENT operations other than tonsillectomy, where the N<sub>2</sub>O:O<sub>2</sub> mixture was 0.65:0.35. The measurements were made during a thyroidectomy procedure.

¶ Surgical operating theatre, where halothane is used at 0.2 vol. %, the N<sub>2</sub>O:O<sub>2</sub> mixture was 0.65:0.35. The measurements were made during a phlebotomy procedure. A sample measured at the level of the head of the anaesthetist, 70 cm from the exhaust port.

\*\* Same operating theatre. A sample measured at the level of the head of the surgeon.

†† Surgical operating theatre, where halothane was used at 0.2 vol. %, the N<sub>2</sub>O:O<sub>2</sub> mixture was 1:1. Measurements were taken during surgery on a polytraumatized patient. A sample measured at the level of the head of the anaesthetist, 70 cm from the exhaust port.

§§ Same operating theatre. A sample measured at the level of the head of the surgeon.

|| || The hall in front of the operating theatres.

¶¶ The personnel resting room.

times the acceptable standard. Halothane concentrations were lower in surgical theatres, where it is used less frequently and at lower concentrations. On the other hand, nitrous oxide time-weighted concentrations in operating theatres, where tonsillectomy in children is performed, was up to 100 times the acceptable standard (Table 1, first line). This concentration was not as high in other theatres, but still exceeded the acceptable standards by approximately 60. Unacceptable concentrations of nitrous oxide were also found in the neighbouring rooms that are not used for operations (Table 1).

Table 2 shows data obtained in the personnel before and after their holidays, and compares them to data in unexposed individuals. The first three lines in Table 2 indicate the red blood cell data; the number of erythrocytes in the blood, the haemoglobin concentration and haematocrit. These three variables were less in the exposed group before the holidays in comparison with control, but not significantly. All three variables were significantly higher after the holidays ( $p < 0.01$  for all), although even then they did not differ from those in the control group.

On the other hand, the number of leukocytes in the blood was significantly lower in the exposed group before the holidays than in the control group ( $p < 0.01$ ), but after the holidays the number of leukocytes increased to become significantly higher than either before the holidays ( $p < 0.01$ ) or in the control group ( $p < 0.05$ ). As compared to the control group, the numbers of segmented neutrophils and monocytes did not decrease significantly during exposure, but increased significantly after the holidays ( $p < 0.05$  for both); eosinophils were not affected at all, but basophils disappeared from the blood during the working period

(Table 2). Compared with the control group, the lymphocytes count was significantly lower in the exposed group before the holidays ( $p < 0.05$ ), whereas it recovered after the holidays and did not differ from that in the control group. The differences did not include the differential count of lymphocytes.

Although percentages of cells bearing a pan-T cell marker (CD2) and CD4 marker were significantly higher during the working period ( $p < 0.01$  for both), neither the numbers of lymphocyte cell subsets bearing T-cell markers CD2, CD4 and CD8 nor the CD4/CD8 cell ratio differed between the control and the exposed groups before vacation; they were therefore not retested after the holidays. However, in comparison to the control group, the CD20 B-cell marker-bearing cells were significantly less frequent in the exposed group, both before ( $p < 0.01$ ) and after the holidays ( $p < 0.05$ ). Similarly, both the percentage and number of NKH-1 marker-positive cells were decreased during the working season ( $p < 0.05$  and  $0.01$ , respectively), but recovered after the holidays, when they were significantly higher than before the holidays ( $p < 0.01$  for both) but did not differ from the control group findings.

In comparison to data obtained in the control group, the NK-cell activity at all the effector:target cell ratios tested tended to be lower in the exposed group during exposition and higher after the holidays (not statistically significant).

The extent of <sup>3</sup>H-thymidine incorporation of lymphocytes after mitogenic stimulation with PHA, Con A and PWM was significantly higher in the exposed subjects during exposure than in the control group ( $p < 0.01$ ,  $p < 0.05$ , and  $p < 0.01$ , respectively); however, SI-values did not differ at all.

**Table 2.** Immunohaematological findings among anaesthetic personnel before and after the holidays.\*

Finding (SD) in exposed personnel									
Variable (measure)	Healthy subjects		p <	Before holiday		p <	After holiday		p < †
Erythrocytes (no. × 10 <sup>12</sup> /litres)	4.47	(0.51)	NS	4.37	(0.31)	0.01	4.73	(0.43)	NS
Haemoglobin (g/litre)	137.6	(15.4)	NS	135.4	(11.7)	0.01	145.2	(9.7)	NS
Haematocrit	0.42	(0.05)	NS	0.41	(0.03)	0.01	0.44	(0.3)	NS
Leukocytes (no. × 10 <sup>9</sup> /litre)	6.49	(1.65)	0.01	5.31	(1.16)	0.01	7.55	(1.46)	0.05
Neutrophils‡ %	58.7	(10.8)	NS	61.6	(7.9)	NS	60.6	(6.5)	NS
number	3.87	(1.42)	NS	3.27	(0.83)	0.01	4.56	(0.98)	NS
Nonsegmented neutrophils %	2.06	(2.13)	0.01	0.86	(1.27)	0.04	1.12	(1.17)	NS
number	0.14	(0.17)	0.04	0.04	(0.08)	NS	0.09	(0.1)	NS
Segmented neutrophils %	56.7	(10.1)	NS	60.8	(7.9)	NS	59.5	(6.7)	NS
number	3.73	(1.33)	NS	3.22	(0.8)	0.05	4.48	(0.94)	0.05
Eosinophils %	2.54	(2.12)	NS	2.29	(1.42)	NS	1.35	(1.62)	NS
number	0.16	(0.14)	NS	0.12	(0.08)	NS	0.11	(0.14)	NS
Basophils %	0.17	(0.38)	0.05	0.0		0.05	0.06	(0.24)	NS
number	0.01	(0.03)	0.03	0.0		0.05	0.004	(0.02)	NS
Monocytes %	2.97	(2.26)	NS	3.24	(1.61)	NS	4.29	(2.42)	NS
number	0.19	(0.16)	NS	0.17	(0.1)	0.05	2.54	(0.58)	NS
Lymphocytes %	35.6	(10.2)	NS	32.9	(7.62)	NS	33.7	(5.6)	NS
number	2.25	(0.68)	0.05	1.75	(0.53)	0.05	2.54	(0.58)	NS
CD2 (T11) %	64.9	(11.8)	0.01	75.8	(11.7)		ND§		
number	1.337	(0.421)	NS	1.324	(0.46)				
CD4 (T4) %	34.9	(9.3)	0.01	43.1	(8.8)		ND		
number	0.741	(0.285)	NS	0.77	(0.328)				
CD8 (T8) %	24.3	(5.7)	NS	26.3	(8.8)		ND		
number	0.513	(0.212)	NS	0.454	(0.188)				
CD4/CD8	1.57	(0.76)	NS	1.86	(0.77)		ND		
CD20 (B1) %	6.88	(3.68)	0.01	3.86	(1.71)	NS	3.73	(3.16)	0.01
number	0.105	(0.086)	0.01	0.066	(0.029)	NS	0.089	(0.073)	0.05
NKH-1 (NK-cells) %	8.96	(7.99)	0.05	4.22	(2.84)	0.01	7.46	(3.51)	NS
number	0.199	(0.176)	0.01	0.069	(0.05)	0.01	0.183	(0.088)	NS
NK-activity at effector : target cell ratio (%) 6 : 1	6.41	(3.08)	NS	5.37	(3.1)	NS	8.22	(5.46)	NS
12.5 : 1	10.77	(5.92)	NS	9.45	(6.4)	NS	13.78	(8.07)	NS
25 : 1	18.47	(7.8)	NS	16.73	(7.21)	NS	21.5	(11.79)	NS
50 : 1	27.01	(11.66)	NS	24.13	(9.89)	NS	28.72	(12.38)	NS
Proliferation to mitogens   <i>PHA</i> cpm (× 10 <sup>3</sup> )	139.0	(34.8)	0.01	173.8	(42.6)		ND		
SI	313.6	(167.9)	NS	279.0	(113.9)		ND		
<i>Con A</i> cpm (× 10 <sup>3</sup> )	81.5	(24.8)	0.05	103.9	(39.5)		ND		
SI	182.8	(108.4)	NS	165.3	(82.3)		ND		
<i>PWM</i> cpm (× 10 <sup>3</sup> )	37.7	(14.3)	0.01	54.8	(24.4)		ND		
SI	91.7	(58.4)	NS	87.2	(48.2)		ND		
Immunoglobulins IgM (mg/litre)	1209.6	(383.7)	NS	1104.3	(472.3)		ND		
IgG (mg/litre)	13150.0	(3106.7)	NS	11979.0	(2292.7)		ND		
IgD (U/ml)	26.5	(26.4)	NS	30.5	(34.3)		ND		
IgA (mg/litre)	1805.0	(994.1)	NS	1599.1	(737.0)		ND		
Phagocytosis (no. of yeast cells/polymorph)	3.12	(0.31)	NS	3.03	(0.37)		ND		

\*Testing was done during April and May 1988; holidays were taken during July and August and lasted 3–4 weeks. Second testing was done immediately after reporting to work.

† In comparison to healthy subjects.

‡ Percent (%) denotes the percentage of the subpopulation of polymorphonuclear leukocytes among the total cell population; the number denotes the respective cell concentration in the blood ( $\times 10^9$ /litre) obtained with the white blood cell differential, and the percentage of a given subpopulation of cells within the lymphocytic flow-cytometric gate.

§Not done.

||PHA, phytohaemagglutinin; Con A, concavalin A, PWM, pokeweed mitogen. cpm is a difference in the number of counts in cultures with and without a mitogen; SI (stimulation index) was obtained by dividing cpm in cultures with a mitogen with cpm in cultures without it.



Serum immunoglobulin concentrations and the extent of yeast cell phagocytosis by blood granulocytes were not affected during exposure to anaesthetics (Table 2).

### Discussion

The concentrations of halothane and nitrous oxide detected in this study were many times higher than the maximum allowed (Table 1, references 32 and 33). Such findings are not exceptional in operating theatres without scavenging devices;<sup>5,34-36</sup> however, the finding prompted us to perform a unique study of the effects of an excessive exposure to these anaesthetics on the human immunohaematological system. At properly protected workplaces, such changes do not occur<sup>37</sup> or are minimal.<sup>36</sup>

Our study has an advantage over those performed in patients exposed to anaesthetics during surgical procedures,<sup>38-41</sup> inasmuch as it circumvented the stress, bleeding and transfusions that accompany surgical interventions and can seriously affect immunohaematological variables, thus masking the effects of the anaesthetics studied. Furthermore, we studied a long-term, heavy exposure and recovery after 4-week holidays, which allowed us to assess reversibility of the changes observed during the working season (Table 2, right column).

The results listed in Table 2 could be divided into several groups. Red blood cell (RBC)-related variables (number of RBC in blood, haemoglobin concentration and haematocrit) were not changed significantly during the exposure, but all of them increased after the holidays (Table 2, first three lines). On the other hand, white blood cells (WBC) responded vigorously to both the anaesthetic and their cessation: during exposure, their number was significantly lower than in the control subjects and increased after the holidays to a significantly higher level than either before the holidays or in the control group. This agrees with data obtained in rats exposed to extremely high nitrous oxide concentrations,<sup>42,43</sup> but, interestingly, is at variance with the absence of immunohaematological changes in rats intermittently exposed to a low concentration of nitrous oxide.<sup>44</sup> Monocytes were unique in their recovery after the holidays. The significance of the profound depletion and inadequate recovery of basophils is doubtful because of their very low proportion among the WBCs (Table 2). Changes in the percentages of WBC subpopulations were much fewer than changes in the respective cell concentrations in the blood; this means that the cell subpopulations oscillated in the same manner.

The effects of anaesthetics on lymphocyte subpopulations has not been studied so far *in vivo*, but only *in vitro*.<sup>45</sup> In the present study, T lymphocytes and their subpopulations appeared less affected than B lymphocytes (Table 2). The percentage among lymphocytes and the concentration of B lymphocytes were the only values that did not recover after cessation of the exposure. Inasmuch as nitrous oxide interferes with DNA synthesis,<sup>45,46</sup> the higher sensitivity of B than of T lymphocytes might be due to a faster turnover of B lymphocytes.

The percentage of NK cells among lymphocytes and their concentration in the blood decreased significantly during exposure to the anaesthetics, but, in contrast to B lymphocytes, they recovered during the holidays. No rebound response was observed (Table 2). This distinguishes NK cells from other mononuclear cell popu-

lations: monocytes and T lymphocytes that were not affected by exposure, total lymphocytes that increased, and B lymphocytes that did not recover (Table 2).

The NK cell activity was not significantly affected either by the exposure or by its cessation (Table 2). The activity was somewhat lower during exposure; however, although consistent, this should probably be interpreted as the lack of influence of nitrous oxide and halothane on NK cell activity. Similar observations were described by others after *in vitro* exposure of peripheral blood mononuclear cells to these anaesthetics.<sup>47,48</sup>

Alteration of *in vitro* response of lymphocytes to the three mitogens used (PHA, Con A, PWM, Table 2), were consistent but difficult to interpret. The uptake of <sup>3</sup>H-thymidine was significantly increased during the period of exposure, but the ratio of the uptake in nonstimulated cultures tended to be lower. Although rather challenging, a suggestion that the exposure to the anaesthetics had a stronger mitogenic influence on resting cells than on stimulated ones, would be premature with the data in hand. Other authors, who studied the influence of human exposure to anaesthetics on the mitogenic stimulation of their lymphocytes, reported similarly inconclusive data.<sup>49</sup> The test sensitivity is probably inadequate to reveal slight immunological changes and differences. If true, however, the observed stimulation of lymphocytes would reveal a difference in the effects of the anaesthetics studied on the immature (inhibition of cell division) and mature (stimulation) cells. A stronger depression of the number than of the activity of NK cells (Table 2) supports this line of reasoning.

Except for IgD, serum immunoglobulin concentrations in the exposed subjects tended to be lower after a long-term exposure, but the differences were not significant (Table 2). Since the serum immunoglobulin concentrations do not correlate with blood concentrations of B lymphocytes (which was significantly decreased both before and after the holidays, Table 2), and testing was not performed after the holidays, interpretation of the findings should await further investigation.

Phagocytic activity of peripheral blood granulocytes was unaltered in the exposed group (Table 2, last line). This generally agrees with the findings of minimal effects of halothane and particularly nitrous oxide on granulocyte functions after an *in vitro* exposure.<sup>22-28,50,51</sup>

Within the limitations of this study, it is difficult to discuss the relative influence of halothane and nitrous oxide on the immunohaematological changes. However, as the latter was used in all surgical procedures, in all theatres, and at higher concentrations than halothane (Table 1), we would put more weight on nitrous oxide. The inhibition of cell growth by nitrous oxide has been attributed to its interference with DNA synthesis through vitamin B<sub>12</sub> inactivation.<sup>45,46</sup> The other factors (X ray, acute stress reaction) which can have influence on our results were excluded.

In summary, our study revealed numerous immunohaematological changes in humans exposed to concentrations of halothane and nitrous oxide several times higher than those suggested as maximally tolerable.<sup>32,33</sup> Most prominent were the changes in WBC, both polymorphonuclear and mononuclear. NK cells and B lymphocytes were considerably more strongly affected than T lymphocytes; B lymphocytes appeared to require a much longer period of

time to recover than NK cells (more than 4 weeks). The influence of the anaesthetics on RBCs were revealed by their significant increase only after cessation of exposure. The effects of NK cell activity, lymphocyte response to mitogens and serum immunoglobulin concentrations remained unclear. Phagocytosis by granulocytes was unaffected. These findings were in agreement with data published elsewhere.

### Acknowledgments

We thank the personnel of the Department of Anaesthesiology and Intensive Care, Dr Mladen Stojanovic Clinical Hospital (Zagreb) for cooperation in the study, and Mr Zvonko Habus of the Quality and Quantity Superintendence Organization in Zagreb for determination of gas and vapour concentrations in the operating theatres.

### References

- BRUCE DL, EIDE KA, LINDE HW, ECKENHOFF JE. Causes of death among anesthesiologists: a 20-year survey. *Anesthesiology* 1968; **29**: 565-9.
- WHITCHER CE, COHEN EN, TRUDELL JR. Chronic exposure to anesthetic gases in the operating room. *Anesthesiology* 1971; **35**: 348-53.
- KNILL-JONES RP, RODRIGUES LV, MOIR DD, SPENCE AA. Anaesthetic practice and pregnancy. Controlled survey of women anaesthetists in the United Kingdom. *Lancet* 1972; **2**: 1326-8.
- BRUCE DL, EIDE KA, SMITH NJ, SELTZER F, DYKES MHM. A prospective survey of anesthesiologist mortality, 1967-1971. *Anesthesiology* 1974; **41**: 71-4.
- American Society of Anesthesiologists ad hoc Committee. Occupational disease among operating room personnel: a national study. *Anesthesiology* 1974; **41**: 321-40.
- WALTS LF, FORSYTHE AB, MOORE JG. Critique: occupational disease among operating room personnel. *Anesthesiology* 1975; **42**: 608-11.
- KNILL-JONES RP, NEWMAN BJ, SPENCE AA. Anaesthetic practice and pregnancy. Controlled survey of male anaesthetists in the United Kingdom. *Lancet* 1975; **2**: 807-9.
- FERSTANDIG LL. Trace concentrations of anesthetic gases: a critical review of their disease potential. *Anesthesia and Analgesia Current Researches* 1978; **57**: 328-45.
- VESSEY, MP. Epidemiological studies of the occupational hazards of anaesthesia—a review. *Anaesthesia* 1978; **33**: 430-8.
- DAVENPORT HT, HALSEY MJ, WARDLEY-SMITH B, BATEMAN PE. Occupational exposure to anaesthetics in 20 hospitals. *Anaesthesia* 1980; **35**: 354-9.
- COHEN EN, GIFT HC, BROWN BW, GREENFIELD W, WU ML, JONES TW, WHITCHER CE, DRISCOLL EJ, BRODSKY JB. Occupational disease in dentistry and chronic exposure to trace anesthetic gases. *Journal of the American Dental Association* 1980; **101**: 21-31.
- BERNOW J, BJÖRDAL J, WIKLUND KE. Pollution of delivery ward air by nitrous oxide. Effects of various modes of room ventilation, excess and close scavenging. *Acta Anaesthesiologica Scandinavica* 1984; **28**: 119-23.
- BROWN BR JR, GANDOLFI AJ. Adverse effects of volatile anaesthetics. *British Journal of Anaesthesia* 1987; **59**: 14-23.
- BRUCE DL, WINGARD DW. Anaesthesia and the immune response. *Anesthesiology* 1971; **34**: 271-82.
- DUNCAN PG, CULLEN BF. Anaesthesia and immunology. *Anesthesiology* 1976; **45**: 522-38.
- BRODSKY JB, COHEN EN. Adverse effects of nitrous oxide. *Medical Toxicology* 1986; **1**: 362-74.
- PARBROOK GD. Leucopenic effects of prolonged nitrous oxide treatment. *British Journal of Anaesthesia* 1967; **39**: 119-27.
- BRUCE DL, WINGARD DW. The effects anaesthesia on transplant rejection. *Anesthesiology* 1969; **30**: 235-6.
- CULLEN BF, DUNCAN PG, RAY-KEIL L. Inhibition of cell-mediated cytotoxicity by halothane and nitrous oxide. *Anesthesiology* 1976; **44**: 386-90.
- BRUCE DL. Halothane action on lymphocytes does not involve cyclic AMP. *Anesthesiology* 1976; **44**: 151-6.
- KUMAR S, TAYLOR G. Effect of surgery on lymphocytotoxicity against tumour cells. *Lancet* 1974; **2**: 1564-5.
- NUNN JF, STURROCK JE, JONES AJ, O'MORÁIN C, SEGAL AW, COADE SB, DORLING J, WALKER D. Halothane does not inhibit human neutrophil function in vitro. *British Journal of Anaesthesia* 1979; **51**: 1101-8.
- NUNN JF, O'MORÁIN C. Nitrous oxide decreases motility of human neutrophils in vitro. *Anesthesiology* 1982; **56**: 45-8.
- DUNCAN PG, CULLEN BF. Neutrophil chemotaxis and anaesthesia. *British Journal of Anaesthesia* 1977; **49**: 345-9.
- BARDOSI L, TEKES M. Impaired metabolic activity of phagocytic cells after anaesthesia and surgery. *British Journal of Anaesthesia* 1985; **57**: 520-3.
- HILL GE, ENGLISH JB, STANLEY TH, KAWAMURA R, LOESER EA, HILL HR. Nitrous oxide and neutrophil chemotaxis in man. *British Journal of Anaesthesia* 1978; **50**: 555-8.
- HILL GE, STANLEY TH, LUNN JK, LIU WS, ENGLISH JB, LOESER EA, KAWAMURA R, SENTKER CR, HILL HR. Neutrophil chemotaxis during halothane and halothane-N<sub>2</sub>O anesthesia in man. *Anesthesia and Analgesia Current Researches* 1977; **56**: 696-702.
- CULLEN BF. The effect of halothane and nitrous oxide on phagocytosis and human leukocyte metabolism. *Anesthesia and Analgesia Current Researches* 1974; **53**: 531-5.
- PALMBLAD J. Stress and immunologic competence: studies in man. In: ADER R, ed. *Psychoneuroimmunology*. New York: Academic Press, 1981: 229-55.
- GABRILOVAC J, ZUPANOVIC Z, OSMAK M. NK-aktivnost u perfernoj krvi zdravih odraslih ljudi. *Radiologica Jugoslavica* 1983; **17**: 327-30.
- PATTERSON-DELAFIELD J, LEHRER RI. A simple microscopic method for identifying and quantitating phagocytic cells in vitro. *Journal of Immunological Methods* 1977; **18**: 377-9.
- Deutsche Forschungsgemeinschaft-Bonn, Maximal Zulässige Konzentrationen auf dem Arbeitsplatz und biologische Toleranzwerte. BRD Makwert, 1984.
- NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH. Occupational Exposure to Waste Anesthetic Gases and Vapors. Criteria for a Recommended Standard. Washington: US Gort. Pr. Off. 1977. DHEW (NIOSH) 77-140.
- DAVENPORT HT, HALSEY MJ, WARDLEY-SMITH B, BATEMAN PE. Occupational exposure to anaesthetics in 20 hospitals. *Anaesthesia* 1980; **35**: 354-9.
- BERNOW J, BJÖRDAL J, WIKLUND KE. Pollution of delivery ward air by nitrous oxide. Effects of various modes of room ventilation, excess and close scavenging. *Acta Anaesthesiologica Scandinavica* 1984; **28**: 119-23.
- SWEENEY B, BINGHAM RM, AMOS RJ, PETTY AC, COLE P. Toxicity of bone marrow in dentists exposed to nitrous oxide. *British Medical Journal* 1985; **291**: 567-9.
- SALO M, RAJAMAKI A, NIKOSKELAINEN J. Absence of signs of vitamin B<sub>12</sub>-nitrous oxide interaction in operating theatre personnel. *Acta Anaesthesiologica Scandinavica* 1984; **28**: 106-8.
- AMESS JAL, BURMAN JF, REES GM, NANCEKIEVILL DG, MOLLIN DL. Megaloblastic haemopoiesis in patients receiving nitrous oxide. *Lancet* 1978; **2**: 339-42.
- O'SULLIVAN H, JENNINGS F, WARD K, MCCANN S, SCOTT JM, WEIR DG. Human bone marrow biochemical function and megaloblastic hematopoiesis after nitrous oxide anesthesia. *Anesthesiology* 1981; **55**: 645-9.
- AMOS RJ, AMESS JAL, NANCEKIEVILL DG, REES GM. Prevention of nitrous oxide-induced megaloblastic changes in bone marrow using folinic acid. *British Journal of Anaesthesia* 1984; **56**: 103-7.
- NUNN JF, CHANARIN I, TANNER AG, OWEN ERTC. Megaloblastic bone marrow changes after repeated nitrous oxide anaesthesia. Reversal with folinic acid. *British Journal of Anaesthesia* 1986; **58**: 1469-70.
- KRIPKE BJ, TALARICO L, SHAH NK, KELMAN AD. Hematologic reaction to prolonged exposure to nitrous oxide. *Anesthesiology* 1977; **47**: 342-8.
- JOHNSON MC, SWARTZ HM, DONATI RM. Hematologic alterations produced by nitrous oxide. *Anesthesiology* 1971; **34**: 42-9.
- CLEATON-JONES P, AUSTIN JC, BANKS D, VIEIRA E, KAGAN E.

- Effect of intermittent exposure to a low concentration of nitrous oxide on haemopoiesis in rats. *British Journal of Anaesthesia* 1977; **49**: 223-6.
45. KANO Y, SAKAMOTO S, SAKURAYA K, KUBOTA T, KASAHARA T, HIDA K, SUDA K, TAKAKU F. Effects of nitrous oxide on human cell lines. *Cancer Research* 1983; **43**: 1493-6.
46. NUNN JF. Clinical aspects of the interaction between nitrous oxide and vitamin B<sub>12</sub>. *British Journal of Anaesthesia* 1987; **59**: 3-13.
47. GRIFFITH CDM, KAMATH MB. Effect of halothane and nitrous oxide anaesthesia on natural killer lymphocytes from patients with benign and malignant breast disease. *British Journal of Anaesthesia* 1986; **58**: 540-3.
48. WOODS GM, GRIFFITHS DM. Reversible inhibition of natural killer cell activity by volatile anaesthetic agents *in vitro*. *British Journal of Anaesthesia* 1986; **58**: 535-9.
49. KOENIG A, KOENIG UD, HEICAPPEL R, STOECKEL H. Differences in lymphocyte mitogenic stimulation pattern depending on anaesthesia and operative trauma: I. Halothane-nitrous oxide anaesthesia. *European Journal of Anaesthesiology* 1987; **4**: 17-24.
50. WELCH WD, ZACCARI J. Effect of halothane and N<sub>2</sub>O on the oxidative activity of human neutrophils. *Anesthesiology* 1982; **57**: 172-6.
51. WELCH WD. Effect on enflurane, isoflurane, and nitrous oxide on the microbicidal activity of human polymorphonuclear leukocytes. *Anesthesiology* 1984; **61**: 188-92.



## Morphine compared with diamorphine

### A comparison of dose requirements and side-effects after hip surgery

S. L. ROBINSON, D. J. ROWBOTHAM AND G. SMITH

#### Summary

*The dose requirements and side effects of morphine were compared with those of diamorphine administered by patient-controlled analgesia in 40 patients following elective total hip replacement. Patients were allocated randomly to receive in a double-blind manner either morphine or diamorphine for postoperative pain relief. There were no significant differences between the two groups with regard to postoperative sedation, nausea, well-being, pain relief and requirements for antiemetic drugs. The dose requirement for diamorphine was approximately 50% of that for morphine.*

#### Keywords

*Analgesia; postoperative.*

*Analgesics; diamorphine, morphine.*

*Equipment; patient-controlled analgesia.*

It is frequently stated that diamorphine causes less nausea, vomiting and sedation and more euphoria than morphine,<sup>1</sup> although it is difficult to find convincing data to support this contention. It has been reported also that diamorphine is twice as potent as morphine.<sup>2,3</sup>

Morphine has been accepted as the standard potent analgesic for many years whereas diamorphine achieved popularity in medical practice only during the first half of this century. It was preferred to morphine because of its greater potency and more rapid onset of action. Increasing illegal use led to its total ban for medicinal use in the USA in 1924. Diamorphine is often more popular than morphine where it is still available, in the UK for example, particularly in the relief of terminal pain and coronary care.

This study was designed to compare the relative potency and side effects of morphine and diamorphine administered for pain relief after total hip replacement via a patient-controlled analgesia system (PCAS).

#### Methods

Forty patients, ASA grades 1–2, aged 44–80 years, undergoing total hip replacement were studied. All patients gave written informed consent to the study which was approved by the local Ethics Committee. Patients who were receiving opioid, sedative or antidepressant therapy and patients undergoing repeat surgery were not studied.

Patients were instructed in the use of a patient-controlled analgesia machine (Graseby) at an interview beforehand. The visual linear analogue scoring (VAS) method for assessment of sedation, nausea and well-being was also explained. Each linear analogue comprised a 10 cm unmarked line, the ends of which denoted the extremes of the variables in question, i.e. wide awake, unable to stay awake; no nausea, worst possible nausea; very unhappy, delighted with everything.

All patients were premedicated with temazepam 10–20 mg orally 1 hour before surgery. Anaesthesia was induced with thiopentone 3–4 mg/kg and tracheal intubation was facilitated by the use of a nondepolarising muscle relaxant. The lungs were ventilated with 66% nitrous oxide and a small concentration of a volatile agent in oxygen for maintenance of anaesthesia. Anaesthesia was supplemented with morphine 0.1–0.2 mg/kg. Morphine was administered intravenously in 2 mg increments in the recovery room until pain control was judged to be satisfactory by the anaesthetist.

Patients were allocated randomly on return to the ward to receive morphine or diamorphine by PCAS, which was programmed to deliver morphine 2 mg or diamorphine 1 mg on demand with a lockout interval of 10 minutes for both groups. Operation of the PCAS was recorded by a printer (Hewlett Packard) which registered the time and success of a patient demand. Intramuscular metoclopramide

S.L. Robinson, BA, RGN, RMN, Research Sister, D.J. Rowbotham, MD, MRCP, FFARCSI, FCAnaes, Senior Lecturer, G. Smith, BSc, MD, FCAnaes, Professor, University Department of Anaesthesia, Leicester Royal Infirmary, Leicester LE1 5WW.

Accepted 10 December 1990.

mid 10 mg was given for nausea at the discretion of the nursing staff.

Visual analogue scores for sedation, nausea and well-being were made at 4, 8, 16, 20 and 24 hours after PCA was started. Pain, nausea and well-being was assessed by a verbal three-point scoring system at the end of the 24-hour study period; severe, moderate and slight or none at all, for pain and nausea; miserable, average and happy for well-being.

Data were analysed by unpaired Student's *t*-test, Wilcoxon rank sum test and Chi-squared test with Yates' correction as appropriate. MANOVA for repeated measures was used for visual analogue scores.

### Results

Four patients in the morphine group were excluded from analysis because of protocol violations. Patients were well matched for age, sex and weight (Table 1).

There were no significant differences between the morphine and diamorphine groups with respect to mean doses of morphine administered in the operating theatre and in the recovery room (Table 2). Mean (SD) 24-hour postoperative consumption after return to the ward was 44 (27.1) mg of morphine in the morphine group compared with 20.2 (10.5) mg of diamorphine in the diamorphine group ( $p = 0.004$ ). No patient self-administered the maximum dose of opioid available.

There were no significant differences between the groups in the requirements for metoclopramide ( $p = 0.50$ ; Table 3) or in the VAS for nausea, sedation or well-being ( $p = 0.66$ , 0.78 and 0.52 respectively; Fig. 1).

**Table 1.** Patient data; (mean (SD)). There were no significant differences.

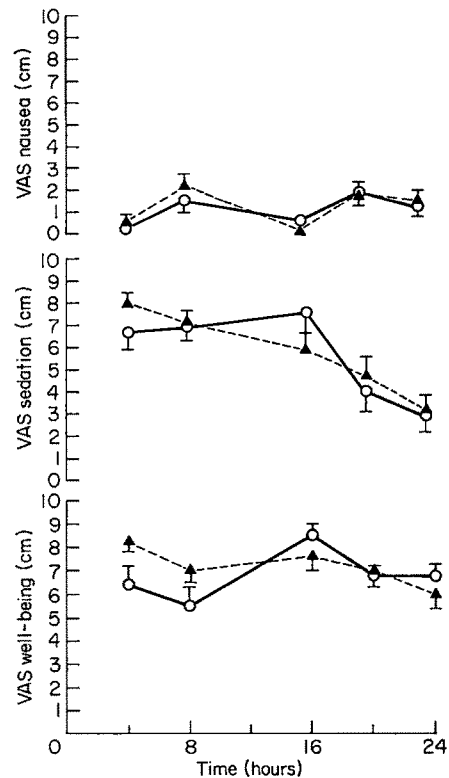
	Morphine group (n=16)	Diamorphine group (n=20)
Sex; m:f	7:9	7:13
Age; years	62.3 (8.4)	58.0 (10.2)
Weight; kg	68.5 (14.0)	66.4 (16.4)
Duration of surgery; hours	1.87 (0.5)	1.95 (0.9)

**Table 2.** Mean (SD) (95% confidence intervals) dose of morphine (mg) given during surgery and in the recovery room. There were no significant differences.

	Morphine group	Diamorphine group	p
Intra-operative	9.3 (2.2) (8.1–10.5)	10.2 (4.2) (8.2–12.2)	0.43
Recovery	2.6 (3.6) (0.6–4.5)	3.5 (3.7) (1.8–5.3)	0.44

**Table 3.** Doses of metoclopramide administered intramuscularly. There were no significant differences ( $p=0.50$ ).

Metoclopramide doses (n)	Morphine group (n)	Diamorphine group (n)
0	10	10
1	3	5
2	2	3
3	0	2
4	1	0



**Fig. 1.** Mean (SEM) Visual analogue scores (VAS) for nausea, sedation and well-being. There were no significant differences  $p = 0.66$ , 0.78 and 0.51 respectively. Morphine,  $\circ$ — $\circ$ ; Diamorphine,  $\blacktriangle$ — $\blacktriangle$ .

The verbal three-point scores (Table 4) for overall experience of pain, nausea and well-being are shown in Table 4. There were no significant differences.

### Discussion

We found no significant differences between the groups over a 24-hour period in the degree of euphoria, nausea and vomiting or sedation, as assessed by subjective visual analogue scores, or in pain relief, nausea and euphoria as assessed by verbal rating. There was, in addition, no significant difference in the requirements for antiemetics between the two groups. Both groups demonstrated relatively low nausea scores. The mean amount of morphine demanded was approximately twice that of diamorphine.

**Table 4.** Results of the three-point verbal scoring system. There were no significant differences.

	Morphine group (n=16)	Diamorphine group (n=20)	p
<b>Pain</b>			0.74
Severe	3	2	
Moderate	9	13	
Slight/none	4	5	
<b>Nausea</b>			0.42
Severe	3	1	
Moderate	3	4	
Slight/none	10	15	
<b>Well-being</b>			0.07
Miserable	1	3	
Average	9	4	
Happy	6	13	

The scientific basis for the alleged superiority of diamorphine over morphine is dubious and since diamorphine is metabolised to monacetyl morphine, and morphine itself, it would be surprising if there were a marked difference. Very few studies authenticate this claim of superiority and the present study is no exception.

Smith and Beecher<sup>4</sup> reported that impairment of mental function, described usually as euphoria, following diamorphine but not morphine was probably caused by the increased speed of onset of action of diamorphine when given in equi-analgesic doses. We found no evidence of increased rate of onset, or of euphoria, compared with morphine. One of the reasons for the popularity of diamorphine amongst drug abusers is its solubility which may account for a previous assumed speed of onset of psychic effects.

Dundee, Loan and Clarke<sup>5</sup> in their study of opioids given as premedication reported no significant difference in the frequency or intensity of subjective effects of diamorphine, but they did report that the effects after diamorphine were of shorter duration (by about 25%) than those of morphine. They found the toxicity of morphine and diamorphine was similar but that morphine caused more emesis than diamorphine and that the latter was more efficacious. It was stressed that these opioids were given only in single doses and that repeated doses may lead to different results. In this study we found no evidence that morphine was associated with more emesis than diamorphine.

Foldes, Swerdlow and Siker<sup>3</sup> estimated that diamorphine was 2–3.3 times more potent than morphine and Dundee and colleagues<sup>5</sup> observed that diamorphine 5 mg was approximately equipotent with morphine 10 mg. Our work, with self-administered intravenous diamorphine and morphine after hip replacement surgery, confirms these observations.

We conclude that, with patient-controlled administration, there is little difference between morphine and diamorphine and there is no evidence to support the view held by many clinicians that diamorphine is superior to morphine in terms of the production of greater euphoria and less nausea and vomiting.

### References

1. SCOTT DB. The use of diamorphine in acute pain. In: SCOTT DB, ed. *Diamorphine its chemistry, pharmacology and clinical use*. New York: Woodhead-Faulkner Limited, 1988; 55–68.
2. JACOBSON L, PHILLIPS PD, HULL CJ, CONACHER ID. Extradural versus intramuscular diamorphine. A controlled study of analgesic and adverse effects in the postoperative period. *Anaesthesia* 1983; **38**: 10–8.
3. FOLDES FF, SWERDLOW M, SIKER ES. *Narcotics and narcotic antagonists*. Springfield, Illinois: Charles C Thomas, 1964.
4. SMITH GM, BEECHER HK. Subjective effects of heroin and morphine in normal subjects. *Journal of Pharmacology and Experimental Therapeutics* 1962; **136**: 47–52.
5. DUNDEE JW, LOAN WB, CLARKE RSJ. Studies of drugs given before anaesthesia. XI diamorphine (heroin) and morphine. *British Journal of Anaesthesia* 1966; **38**: 610–9.

## A double-blind study of the speed of onset of analgesia following intramuscular administration of ketorolac tromethamine in comparison to intramuscular morphine and placebo

A. S. C. RICE, J. LLOYD, C. G. MILLER, R. E. BULLINGHAM AND G. M. O'SULLIVAN

### Summary

A double-blind, randomised, parallel group, placebo-controlled study was performed in 85 patients to compare the speed of onset of analgesia following the intramuscular administration of a single dose of 30 mg of ketorolac tromethamine, 10 mg of morphine or placebo. A new, sensitive, method was used to measure the latency of analgesia. The onset of analgesia was defined by the time taken for the pain intensity score to reach a specified percentage of the baseline value. Twenty-five percent of patients achieving a 50% reduction in baseline pain intensity score appears to be the most appropriate parameter to assess the speed of onset of analgesia of ketorolac and morphine in the postoperative setting. Paired comparison demonstrated that ketorolac had a significantly faster onset of analgesia ( $p = 0.03$ ) when compared to placebo, whilst comparison of morphine to placebo analgesic latency ( $p = 0.06$ ) just failed to reach significance. There was no significant difference between the analgesic onset time of ketorolac and morphine ( $p = 0.73$ ). Intramuscular ketorolac and intramuscular morphine have comparable analgesic onset times in the postoperative pain context. However, the sensitive method of measuring onset of analgesia described, highlights the slow onset of analgesia when analgesics of known efficacy are given by the intramuscular route in the postoperative period. More attention should be given to the speed of onset of analgesia in future assessments of analgesics.

### Key words

Analgesics; morphine.

Anti-inflammatory agents; nonsteroidal.

Pain; measurement.

Ketorolac is a drug which displays potent analgesic, anti-inflammatory and antipyretic properties. The efficacy of ketorolac when given by the intramuscular route in post-operative pain of moderate intensity has been demonstrated.<sup>1–3</sup> This study aimed to compare the onset time of analgesia after intramuscular administration of equipotent<sup>3</sup> doses of ketorolac and morphine. The time of onset of analgesia is generally derived by interpolation of pain scores made at the time of injection and 30 minutes later. The object of this study was to record the pain scores at more frequent intervals and thereby to obtain a more accurate assessment of analgesic latency.

### Methods

Local ethics committee approval was obtained before the start of this double-blind, placebo-controlled, randomised study, with three parallel groups. Pregnant or lactating

females were not studied, nor were patients suffering from peptic ulcers or ulcerative colitis. Patients with significant cardiovascular, renal, hepatic, respiratory or nervous system dysfunction were not considered for entry into the study. Known sensitivity to aspirin or other prostaglandin synthetase inhibiting drugs and the presence of a haemorrhagic diathesis also excluded patients from the study.

On the day before surgery the study was carefully explained to, and written consent was obtained from, 85 patients scheduled to undergo elective surgery. They were instructed on the use of the pain intensity rating scale and tests of cognitive function which included the ability to correctly name the day of the week and the repeated subtraction of 7 from 99.

All patients received an oral premedication of temazepam (20 mg) 2 hours before anaesthesia. Anaesthesia was induced by an intravenous injection of thiopentone (4–5 mg/kg). General anaesthesia was maintained with nitrous oxide, a volatile agent and fentanyl (1.5–2 µg/kg). In the

A.S.C. Rice, FFARCS, Research Fellow, G.M. O'Sullivan, MD, FFARCS, Consultant, Department of Anaesthetics, St. Thomas' Hospital, Lambeth Palace Road, London SE1 7EH, J. Lloyd, MSc, C.G. Miller, PhD, R.E. Bullingham, MSc, FFARCS, Syntex Research, Maidenhead, Berks.

Correspondence to Dr G. O'Sullivan.

Accepted 10 December 1990.



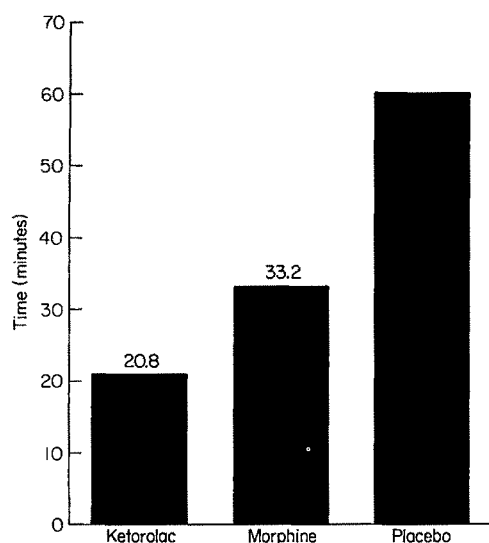


Fig. 1. The time (minutes) after administration of analgesia by which 25% of patients had achieved a 50% reduction in pain intensity. Only 15% of patients in the placebo group achieved a 50% reduction in the pain intensity score in 60 minutes.

cases where nondepolarising muscle relaxants had been used, any residual paralysis at the conclusion of surgery was reversed with neostigmine (0.06 mg/kg) and glycopyrronium (0.0125 mg/kg).

Patients experiencing pain of at least moderate severity within 2 hours of the completion of surgery were allowed to continue in the study. The cognitive function tests were performed before the study started. If satisfactory, patients were then randomly allocated to receive intramuscular ketorolac 30 mg, intramuscular morphine 10 mg or intramuscular placebo; each were identical in appearance and administered in a 1-ml volume. Pain intensity scores on a scale of 0–100 (the extremes representing 'no pain' and 'the worst pain you could possibly imagine') were obtained before injection, every 2 minutes for the first 10 minutes following injection, and every 5 minutes for the next 50 minutes. Rescue analgesia was given if required, but not earlier than 30 minutes after the start of the study, unless the patient's state required earlier treatment, in which case the study was discontinued. Vital signs were measured every 15 minutes from the start of the study. Patients were interviewed at the time of the study and 24 hours later. Possible side effects, complaints elicited by indirect questioning and drug usage were recorded.

In order to calculate the time of onset of analgesia it was necessary to smooth the pain intensity score/time curve so that an accurate estimate could be made. However, it was first necessary to construct a common pain intensity scale for all subjects. This was performed by converting each score into a percentage of the baseline score for each patient. Each data point was then, in turn, replaced by a weighted mean which was derived by using a three-point moving average technique. This takes three consecutive points and calculates their mean, giving most weight to the central point. The technique also allows for differences in elapsed time between each data point. This method removes the minor alterations in the pain intensity score/time curves, but not the underlying trends. The formula used for this calculation is shown in appendix 1.

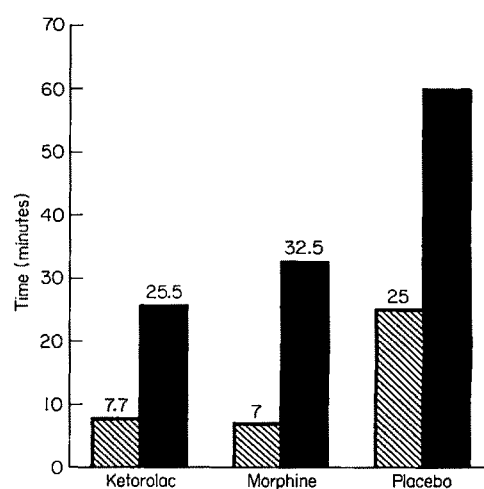


Fig. 2. The time (minutes) after administration of analgesia by which 25% (▨) and 50% (■) of patients had achieved a 25% reduction in pain intensity. Only 46% of patients in the placebo group achieved a 25% reduction in the pain intensity score in 60 minutes.

The time of onset of analgesia was then set as the time at which the smoothed curve first decreased to 25% and 50% of the baseline pain intensity score. The cumulative percentage of patients reaching these criteria as a function of time then gave a frequency curve for onset of analgesia. The time to onset of analgesia and time to the next analgesic were analysed by survival analysis. The results for each treatment group were summarised using the Kaplan-Meier estimator.<sup>4</sup> For analgesic latency and time to next analgesic, groups were compared using the generalised Wilcoxon test (two-tailed with a 5% level of significance).

## Results

Eighty-five patients participated in the trial, 28 each in the morphine (10 male, 18 female) and ketorolac (7 male, 21 female) groups and 29 (10 male, 19 female) in the placebo group. Three patients, all in the placebo group, failed to perform the pain intensity scoring adequately after operation and were excluded from further analysis; this left 26 patients in this group.

The age range was 20 to 77 years with median ages (years) of 40.5 (morphine), 48 (ketorolac) and 43.5 (placebo). The median weights (kg) were 70.3 (morphine), 67.3 (ketorolac) and 72 (placebo), with a range of 48–113.7 kg. The most common operations were abdominal hysterectomy and hernia repair. Together, these accounted for 57% of all operations. The median times for duration of surgery were similar in all groups.

There was little difference between the groups in the prestudy pain intensity scores (mean, SD): ketorolac 59.8 (19.55), morphine 57.7 (20.34) and placebo 62.8 (23.99).

The time by which 25% of patients achieved a 50% reduction in pain intensity score is shown in Figure 1. In no group did 50% of patients achieve a 50% reduction in pain intensity within the 60 minutes of the study. The times by which 25% and 50% of patients achieved a 25% reduction in pain intensity scores are shown in Figure 2.

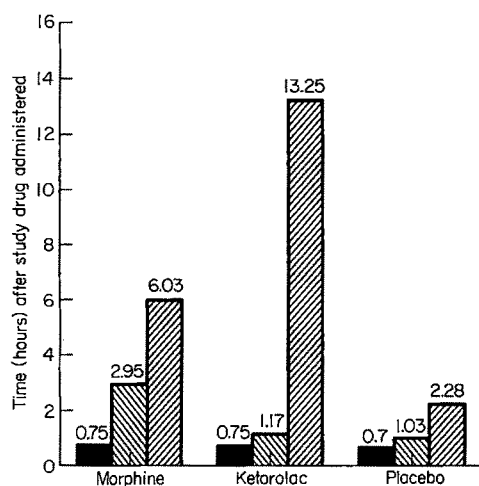


Fig. 3. The time (hours) following administration of study analgesic by which the next analgesic drug was given. This is shown for 25% (■), 50% (▨) and 75% (▩) of patients in each study group.

When a criterion of 25% reduction in pain intensity scores was used to define onset of analgesia (Fig. 2) there was no statistically significant difference between any pairs of groups, neither for 25% nor for 50% of patients to reach this level of analgesia (Wilcoxon test). When a criterion of 50% reduction in pain intensity score was used then in none of the groups did 50% of the patients reach the defined level of analgesia within the 60-minute study period. When a criterion of 25% of patients achieving a 50% reduction in pain intensity score was used to measure analgesic latency (Fig. 1) then a paired comparison (Wilcoxon test) to placebo showed a significant reduction for ketorolac ( $p = 0.03$ ) and a smaller difference for morphine, which just failed to reach significance ( $p = 0.06$ ). There was no difference when the analgesic latency of morphine and ketorolac were compared ( $p = 0.73$ ).

The time from the initial dose of analgesia to the time at which the next analgesic was administered is shown in Figure 3. Ketorolac was superior to morphine, in this respect.

There was no major morbidity in any of the groups. The most frequent adverse event was vomiting, which occurred seven times in the morphine group and six times in each of the other two groups. No patients withdrew from the study because of an adverse event. A total of 29 patients withdrew from the study because of insufficient analgesia: eight in the morphine group, nine in the ketorolac group and 12 in the placebo group.

### Discussion

The time to onset of analgesia is generally derived from the interpolation of pain intensity differences derived from pain intensity scores taken before treatment and at 15 or 30 minutes after injection.<sup>5-7</sup> Linear interpolation between points does not allow for accurate assessment of onset times, because the onset of effect is not necessarily linear. Measures of onset time, as usually quoted, do not compare the value for an analgesic with that for placebo, but refer only to decreases from the baseline within a group. Such an onset time cannot then be claimed as drug related, but

could just be spontaneous regression of pain. Finally, most studies of analgesics are not primarily concerned with onset times and merely obtain this information as secondary data. Few time points are recorded and there is insufficient data to ensure that outlying data points do not distort the derived onset time. Vignoni *et al.*<sup>8</sup> have taken pain scores at 5-minute intervals following drug administration, but this work was hampered by the use of an insensitive three-point pain scoring system.

This study was primarily concerned with the speed of onset of analgesia and therefore a method was designed which could accurately measure this and address the above concerns. The definition of onset time is not straightforward since patients do not have the same pain intensity at the time of analgesic administration and do not all achieve the same degree of reduction in pain from the same dose of an analgesic. Onset of analgesia must therefore specify two parameters to define a time of occurrence; the percentage of patients reaching a satisfactory level of analgesia and the percentage level of pain relief from baseline that a set percentage of patients achieve. If these parameters are set too close to zero then any difference between the groups is masked by the noise of the system. Alternatively, if the criteria are set too high then very few patients will achieve this level and there would be insufficient data to give a meaningful result. The inclusion of a placebo group allowed the validity of the set point parameters to be precisely confirmed, since this determined both the noise of the system and its sensitivity. Frequent measurements using a 100 point scale were used to increase the experimental sensitivity and using this method the speed of onset of analgesia of equipotent doses<sup>3</sup> of intramuscular morphine and ketorolac could be compared.

Fifty percent pain intensity reduction for 50% of patients appears to be too severe a criterion to measure analgesic onset time in this postoperative pain model. However, it could be claimed that 10 mg of morphine is not a sufficiently efficacious analgesic, but this is probably not a valid criticism since the expected degree of side effects from this analgesic dose were seen. Twenty-five percent of patients reaching a 50% reduction in pain intensity seems an appropriate parameter to measure analgesic onset time in the model studied here.

The efficacy of intramuscular ketorolac compared to opioids has been well documented<sup>1-3,9,10</sup> and we have confirmed this; intramuscular ketorolac is at least as efficacious as intramuscular morphine and better than intramuscular placebo. The time to next analgesic data, analysed by survival methods, indicate that there is no difference between ketorolac, morphine and placebo for the time by which the first 25% of patients required the next dose of analgesic. Fifty percent of patients receiving ketorolac required the next analgesic sooner than 50% of those patients receiving morphine, and ketorolac was not superior to placebo in these patients. However, when the time to next analgesic data for 75% of patients is examined it appears that 25% of patients who receive ketorolac experience a much longer period (13.25 hours) before requiring another analgesic than did those receiving morphine or placebo, although morphine was superior to placebo in this respect. The reasons for this long duration of action of ketorolac could include the nature of the surgery performed and a beneficial effect of the use of nonsteroidal anti-inflammatory analgesics in the early post-

operative period, in a susceptible population. Thus ketorolac may compliment morphine when the two are used in combination in the immediate postoperative period.

The side effects experienced by all the groups, including placebo, were similar in frequency and nature, suggesting that none of the drugs shows any particular advantage in this respect.

In this study the time to onset of analgesia of intramuscular ketorolac, morphine and placebo has been measured by a sensitive method and the time to onset was similar for ketorolac and morphine, but both ketorolac and morphine had a faster onset than placebo although this difference only reached statistical significance in the case of ketorolac.

The literature provides little information regarding the onset of analgesia from an intramuscular administered analgesic in the postoperative period. The sensitive method described here reveals the relatively slow onset of a well established analgesic such as morphine and an equipotent dose of a nonsteroidal anti-inflammatory analgesic (ketorolac). This study demonstrates that not only should future studies examine the quality of analgesia in the early postoperative period, but that onset of analgesia should also be measured by a sensitive method as described here.

#### Acknowledgments

We thank Mr R. Taylor of Syntex Research for his invaluable statistical expertise and the recovery ward nursing staff at St. Thomas' Hospital for their cooperation.

#### Appendix 1.

Smoothed pain intensity score at  $T_a$

$$= \frac{[T_{a+1} - T_a] \times P_{a-1} + [T_{a+1} - T_{a-1}] \times P_a + [T_a - T_{a-1}] \times P_{a+1}}{2 \times [T_{a+1} - T_{a-1}]}$$

$T_{a-1}$  Time of preceding data point.

$T_a$  Time of point to be smoothed (middle).

$T_{a+1}$  Time of next data point.

$P_{a-1}$  Preceding pain intensity score.

$P_a$  Pain intensity score to be smoothed (middle).

$P_{a+1}$  Next pain intensity score.

#### References

1. YEE J, BROWN, ALLISON, C, WILDMAN K. Analgesia from intramuscular ketorolac tromethamine compared to morphine (ms) in 'severe' pain following 'major' surgery. *Clinical Pharmacology and Therapeutics* 1985; **37**: 239.
2. YEE J, BRADLEY R, STANSKI D, CHERRY C. A comparison of analgesic efficacy of intramuscular ketorolac tromethamine
3. YEE JP, KOSHIVER JE, ALLBON C, BROWN CR. Comparison of intramuscular ketorolac tromethamine and morphine sulfate for analgesia of pain after major surgery. *Pharmacotherapy* 1986; **6**: 253-61.
4. COX DR, OAKES D, In: *Analysis of survival data*. London: Chapman and Hall, 1984.
5. TIGERSTEDT I, JANHUNEN L, TAMMISTO T. Efficacy of diclofenac in a single prophylactic dose in postoperative pain. *Annals of Clinical Research*. 1987; **19**: 18-22.
6. MARSALA F, CAVRINI P, BUFALINO L, GARDINI, F. Treatment of acute pain of ureteral and biliary colic with naproxen sodium administered by the parenteral route. *International Journal of Clinical Pharmacology Research* 1986; **6**: 495-500.
7. MCQUAY HJ, CARROLL D, POPPLETON P, SUMMERFIELD RJ, MOORE RA. Fluradoline and aspirin for orthopedic postoperative pain. *Clinical Pharmacology and Therapeutics* 1987; **41**: 531-6.
8. VIGNONI A, FIERRO A, MORESCHINI G, CAU M, AGOSTINO A, DANIELE E, FOTI G. Diclofenac sodium in ureteral colic: a double blind comparison trial with placebo. *Journal of International Medical Research* 1983; **11**: 303-7.
9. O'HARA DA, FRAGEN RJ, KINZER M, PEMBERTON D. Intramuscular ketorolac tromethamine as compared to morphine sulfate in postoperative pain. *Anesthesiology* 1986; **65**: A187.
10. FRAGEN RJ. A comparison of intramuscularly injected ketorolac and morphine in postoperative pain. *Clinical Pharmacology and Therapeutics* 1987; **41**: 212.

and meperidine in postoperative pain. *Clinical Pharmacology and Therapeutics* 1986; **39**: 237.

## Intubation through the laryngeal mask

### A technique for unexpected difficult intubation

M. L. HEATH AND J. ALLAGAIN

#### Summary

*Unexpected difficulty with tracheal intubation contributes to anaesthetic mortality. The laryngeal mask can almost always be placed satisfactorily and its position should facilitate blind intubation. A 6-mm cuffed tube will pass through both adult sizes of the mask and this study tested the feasibility of intubation through the mask. The effect of the application of cricoid pressure on the technique was also investigated. Intubation via the laryngeal mask was attempted in 100 routine patients: of the first 50 (group 1, no cricoid pressure), 45 (90%) were successfully intubated. Maintenance of cricoid pressure throughout the manoeuvre (group 2) reduced the success rate significantly to 56% ( $p < 0.05$ ). Despite the possibility that cricoid pressure may have to be interrupted momentarily, the ease with which the technique can be learnt, and the immediate availability of the necessary apparatus suggest that it should be considered for inclusion in failed intubation drill.*

#### Key words

*Intubation, tracheal; cricoid pressure.  
Equipment; laryngeal mask.*

Difficulties with tracheal intubation contribute substantially to anaesthetic morbidity and mortality. Bellhouse and Doré<sup>1</sup> estimate that a minimum of 600 deaths annually within the developed world alone is attributable to such events. Enormous effort<sup>1–7</sup> has been expended on developing systems for predicting difficulty; however, all authors acknowledge that 100% prediction is an unattainable goal. The aim of prediction is to allow appropriate preparations to be made and sophisticated techniques such as fiberoptic intubation to be employed; it is not usually possible to use these when unexpected difficulty is encountered. Emergency cases reduce both the time available for, and the range of, these possibilities, but the likelihood of a full stomach requires that the lungs be protected with minimum risk of aspiration between induction of anaesthesia and placement of the cuffed tracheal tube. Unexpected difficult intubation most commonly presents in obstetric practice, when fetal distress in particular may reduce the time available for alternative measures.

The technique of placement of the laryngeal mask<sup>8</sup> does not require the larynx to be seen. However, in all but a very few cases, it forms a reliable laryngeal seal and provides an extremely clear airway. Fiberoptic studies have shown that its distal aperture normally lies very close to the vocal cords. The value of the laryngeal mask in cases of difficult intubation is well established.<sup>9</sup> It does not, however, reliably protect the patient from regurgitation and aspi-

ration.<sup>10</sup> The use of the laryngeal mask as an aid to difficult intubation has been described.<sup>11</sup> However, the technique suggested employed the airway to place a gum elastic introducer, over which a tracheal tube was railroaded after removal of the airway. Railroading is not always easy or even possible in our experience and the additional time taken can increase the risk of hypoxia and/or regurgitation.

A 6-mm cuffed tracheal tube will pass through sizes 3 and 4 laryngeal mask and this study was undertaken to assess the possibility of using the laryngeal mask as an aid to blind intubation with particular reference to the need to protect the lungs from aspiration of stomach contents.

#### Methods

The study was approved by the District Ethics Committee and each patient gave informed consent. Healthy adult patients (ASA 1 or 2), who presented for elective or scheduled surgery not requiring access to the pharynx and for whom tracheal intubation would form a normal part of the anaesthetic technique, were selected sequentially from the authors' routine operating lists. Patients were premedicated with diazepam 10 mg and anaesthesia was induced intravenously with propofol. The dose was judged clinically and came within the range 1.5–2.5 mg/kg. Lignocaine 20 mg was added to each 20 ml syringe of propofol. Pentazocine 60 mg was given and the patient's lungs ventilated gently

M.L. Heath, MB, BS, FCAnaes, Consultant, J. Allagain, Senior Operating Department Technician, Anaesthetic Department, Lewisham Hospital, Lewisham High Street, Lewisham, London SE13 6LH.

Part of this work was presented to the Anaesthetic Research Society at its meeting held in November 1989.

Accepted 29 September 1990.

for one minute via a facemask and a Bain system with oxygen, to which an inhalation agent was added (halothane 2% or enflurane 3%). The laryngeal mask was then inserted in the normal manner using a size 3 for females and a size 4 for males; the anaesthetic system was connected after the cuff had been inflated with 20–30 ml of air and suxamethonium 1 mg/kg was given. A few more breaths were given and, when muscular relaxation was judged to have occurred, intubation was attempted. The tubes employed were 6 mm internal diameter with a standard cuff, either nasal (Portex) or oral (Mallinkrodt) pattern. They were used as supplied, full length (28.5 cm) with a standard connector; the relative lengths of the laryngeal mask and tracheal tube allow approximately 8 cm of the tube tip to project beyond the laryngeal aperture bars, thus placing the upper border of the cuff about 3 cm below the vocal cords when positioned in the patient. Good lubrication is required and the tube must be rotated 90° to the left during passage. This brings the bevel anteriorly so that the tip passes easily through the aperture bars; the rotation is allowed to correct itself during the last few centimetres of passage. Patients were placed in a normal intubating position with the neck flexed and the head extended. Slight adjustment of the position was made if any resistance to passage was encountered; in no case was force used. The anaesthetic system was then connected to the tracheal tube and lung inflation checked visually and with a stethoscope. The laryngeal mask was taped to the patient's face and the tube and laryngeal mask connectors were taped securely together. For the first group of 25 patients (group 1A), each stage of the procedure was timed by an independent observer; a time limit of one minute was set for placement of the laryngeal mask and passage of the tracheal tube. Pulse oximetry became routinely available in the anaesthetic room during this initial series, and experience showed that neither desaturation nor clinically significant changes in end-tidal CO<sub>2</sub> levels occurred during the procedure. Timing was therefore abandoned and a further 25 patients (group 1B) studied.

To assess the effects of the application of cricoid pressure during the procedure, a further 50 similar patients were studied (group 2). It was not necessary to employ a standard rapid sequence induction since they were fully prepared patients: pre-oxygenation was not used and cricoid pressure was applied only after consciousness was lost. Cricoid pressure was applied in a standard manner by an experienced assistant and maintained during laryngeal mask placement and attempted tracheal intubation. When failure was unequivocal and immediately obvious, cricoid pressure was released and the manoeuvre repeated, if necessary after adjusting slightly the position of the laryngeal mask.

## Results

### Group 1A

Twenty-one out of 25 patients (84%) were intubated; of these, 18 intubations were at the first attempt and the mean time between disconnecting the system from the laryngeal mask and reconnecting it to the tracheal tube was 13 seconds (range 7–27); for the other three, minor adjustments of the patient's position or that of the mask, or withdrawal of some of the air in the cuff were required.

Table 1. Results of first 50 patients.

	<i>n</i>	Intubated (%)	Laryngeal mask not placed in one minute (%)	Intubation abandoned (%)
Group 1A	25	21 (84)	3 (12)	1 (4)
Group 1B	25	24 (96)	0	1 (4)
Total	50	45 (90)	3 (6)	2 (4)

Three attempts were abandoned because the laryngeal mask was not satisfactorily positioned within one minute.

The remaining patient was a heavy smoker who reacted to the intubation attempt with coughing and straining and was therefore intubated conventionally. In retrospect, the initial intubation had almost certainly been successful since her lungs were no easier to inflate after intubation under direct vision.

### Group 1B

Twenty-four out of 25 patients were intubated (96%), 19 at the first attempt and five after minor adjustments. The combined results of groups 1A and 1B show that intubation was successful in 90% of patients (Table 1).

### Group 2

Twenty-eight out of 50 patients (56%) were intubated whilst cricoid pressure was maintained, 21 at the first attempt and seven after minor adjustments. Of the 22 failures, 17 were immediately obvious and a further attempt was made in these patients after release of cricoid pressure; this resulted in success in 15 and failure in two. Of the five in whom a repeat attempt was not made, two patients had recovered from suxamethonium and a further three were equivocal, in that tracheal intubation appeared to have been successful as judged by auscultation and capnometry. Respiration, however, appeared to be somewhat obstructed and it was decided that the tracheal tube should be withdrawn. These results are summarised in Table 2.

### Comparisons between groups 1A and B combined and group 2

The difference between the success rate in group 1 (90%) and group 2 with cricoid pressure (56%) was significant at the 5% level (Chi-squared 3.95,  $p < 0.05$ ); however the final success rate in group 2 patients, including those in whom further attempts were made without cricoid pressure (43 out of 45, 96%), was similar.

There seemed a possibility that a higher proportion of female patients had been successfully intubated. The results for males and females were therefore separated (Table 2) and showed that 12 of the 18 females (66%) and 16 of the 32 males (50%) were intubated with cricoid pressure. This difference is not significant at the 5% level. The final satisfactory intubation rate was 26 out of 32 (81%) for males and 17 out of 18 (94%) for females, a nonsignificant difference.

Table 2. Results of second 50 patients.

	All subjects (%)	Male (%)	Female (%)
Intubated with cricoid pressure	28 (56)	16 (50)	12 (66)
Intubated after release of cricoid pressure	15 (30)	10 (31)	5 (28)
Total intubated	43 (86)	26 (81)	17 (94)
Failed to intubate	2 (4)	2 (6)	0
Not attempted without cricoid pressure	5 (10)	4 (13)	1 (6)
Total	50	32	18

### Discussion

In these unselected patients the technique proved to be easy with a high success rate. However, no assessment was made of the ease with which they could have been intubated conventionally, apart from the fact that three patients (all intubated easily through the laryngeal mask) were noted to have been difficult to intubate in previous anaesthetic records. Seven patients in group 1A were noted to have anterior crowned, capped or loose teeth which might have been endangered by standard laryngoscopy but were unstressed by the guided technique. The 6 mm cuffed tube is the largest that can pass through the current production models of the laryngeal mask. This size is smaller than normally chosen for adult patients (although sometimes resorted to in special or difficult circumstances); furthermore, the necessary length increases the resistance to gas flow. No difficulty was found, however, in maintaining acceptable end-tidal CO<sub>2</sub> levels with controlled ventilation, which is in line with our experience in using long, 5 mm tubes for microlaryngoscopy. The majority of patients breathed adequately spontaneously but it should be borne in mind that most of them were placed in a head-up position, were healthy and were given an anaesthetic deliberately designed to minimise respiratory depression. The three patients in group 2 who were regarded as unsatisfactorily intubated, despite clear evidence of the tube being placed within the respiratory pathway, had probably suffered epiglottic downfolding,<sup>12</sup> encouraged by cricoid pressure. All were male and were noted to have large, floppy epiglottides, as were the two patients in whom intubation failed, both with and without cricoid pressure.

There would appear to be three situations in which this technique may prove to be of value. First, where fragile teeth or restorations prove to be at risk during routine laryngoscopy the technique may be preferred. Second, in anticipated difficult (but not impossible) conventional intubation in the prepared patient, the technique may be tried before progressing to more traumatic or complex manoeuvres, with all equipment prepared and checked before starting. Thirdly, and more controversially, the technique may be considered where unexpected difficulty is experienced in a patient at risk from regurgitation (in whom therefore the laryngeal mask alone is not a completely satisfactory option). The advantages include the ease with which the technique may be learnt, the immediate availability of the necessary equipment, the rapid initial control of oxygenation when the laryngeal mask is inserted and the short time normally required for intubation, even if cricoid pressure needs to be relaxed for a moment. These features are not so obviously applicable to a recently advocated radiological solution to the problem<sup>13</sup> nor to the 'divided airway' of the 1940s recommended in recent corre-

spondence.<sup>14</sup> On the basis of our admittedly limited experience we would suggest that momentary relaxation of cricoid pressure during placement of the mask and again during the intubation manoeuvre will provide the best compromise between protection against regurgitation during the procedure and rapid successful intubation. There is a trend towards a higher success rate in females and this may reassure anaesthetists contemplating its use for emergency Caesarean section where hypoxia and delay may further compromise the fetus. Reports of intubation difficulties solved by laryngeal mask placement are no rarity<sup>15</sup>; it would be most helpful in future if anaesthetists finding themselves in this situation would try to intubate in the manner described, in order to assess the usefulness of the technique in more demanding circumstances. The technique described in this paper was developed intuitively; there is clearly scope for identifying strategies to improve the success rate. The use of a fibroscope seems an obvious development; not only would it aid the anaesthetist in difficult but unhurried circumstances, but it could allow refinement of the technique for use in situations where either the equipment or time to use it are not available. Further studies are planned to assess the cardiovascular responses to this method of tracheal intubation.

### References

- BELLHOUSE CP, DORÉ C. Criteria for estimating likelihood of difficulty of endotracheal intubation with the Macintosh laryngoscope. *Anaesthesia and Intensive Care* 1988; **16**: 329-37.
- WILSON ME, SPIEGELHALTER D, ROBERTSON JA, LESSER P. Predicting difficult intubation *British Journal of Anaesthesia* 1988; **61**: 211-6.
- SAMSOON GL, YOUNG JRB. Difficult tracheal intubation: a retrospective study. *Anaesthesia* 1987; **42**: 487-90.
- MALLAMPATI SR, GATT SP, GUGINO LD, DESAI SP, WARAKSA B, FREIBERGER D, LIU PL. A clinical sign to predict difficult intubation: a prospective study. *Canadian Anaesthetists' Society Journal* 1985; **32**: 429-34.
- HORTON WA, FAHY L, CHARTERS P. Disposition of cervical vertebrae, atlanto-axial joint, hyoid and mandible during x-ray laryngoscopy. *British Journal of Anaesthesia* 1989; **63**: 435-8.
- WHITE A, KANDER PL. Anatomical factors in difficult direct laryngoscopy. *British Journal of Anaesthesia* 1975; **47**: 468-74.
- FAHY L, HORTON WA, SPRIGGE JS, CHARTERS P. X-ray laryngoscopy in patients with a history of difficult laryngoscopy during pregnancy. *British Journal of Anaesthesia* 1989; **62**: 234P.
- BRAIN AIJ. *The Intavent laryngeal mask: instruction manual*. Reading: Brain Medical, 1990.
- BRAIN AIJ. Three cases of difficult intubation overcome by the laryngeal mask airway. *Anaesthesia* 1985; **40**: 353-5.
- CYNA AM, MACLEOD DM. The laryngeal mask: cautionary tales. *Anaesthesia* 1990; **45**: 167.
- CHADD GD, ACKERS JW, BAILEY PM. Difficult intubation aided by the laryngeal mask airway. *Anaesthesia* 1989; **44**: 1015.

12. PAYNE J. The use of the fiberoptic laryngoscope to confirm the position of the laryngeal mask. *Anaesthesia* 1989; **44**: 865.
13. INNOCENTE F, ORI C, GIRON GP. Tracheal intubation under fluoroscopic control. X ray-guided orotracheal intubation in three cases of impossible direct laryngoscopy. *Anaesthesia* 1990; **45**: 675-7.
14. WELSH BE, Aid to intubation. *British Journal of Anaesthesia* 1989; **62**: 469-70.
15. MCCLUNE S, REGAN M, MOORE J. Laryngeal mask airway for Caesarean section. *Anaesthesia* 1990; **45**: 227-8.



## Epidural bupivacaine dilution for labour

A comparison of three concentrations infused with a fixed dose of fentanyl

H. A. NOBLE, G. R. ENEVER AND T. A. THOMAS

### Summary

We have compared the effects of three epidural infusions in a randomised, double-blind study of 56 primigravid mothers in labour. An initial dose of bupivacaine 0.5% 8 ml was followed by infusion of either bupivacaine 0.125%, 0.062% or 0.031%. All solutions contained fentanyl 0.0002% and were run at 7.5 ml/hour. Women receiving the most dilute solution had significantly less analgesia ( $p < 0.001$ ) for the first 4 hours of the study, but pain scores were then similar for the three groups. No obvious benefit was gained by using very dilute bupivacaine.

### Key words

Anaesthesia; obstetric.

Anaesthetic techniques, regional; epidural infusion.

Continuous epidural infusions are being used increasingly for analgesia during labour, and have been shown to be safe, effective, and to offer advantages over intermittent techniques.<sup>1–3</sup> The ideal solution for use by infusion has yet to be defined. It should provide good analgesia without producing excessive sensory or motor blockade. This should allow the best chance of a normal vaginal delivery while minimising systemic toxicity and side effects. A combination of local anaesthetic agents with opioid analgesics seems to come closest to the ideal and has been widely investigated in recent years.<sup>4</sup> Unfortunately, epidural opioids have many side effects,<sup>5</sup> including respiratory depression, nausea and pruritus. They seem to be related to the use of large doses, either by bolus or infusion. It would therefore seem sensible to use as little opioid as necessary.

Our recent study using a moderately dilute solution of bupivacaine, 0.125%, combined with fentanyl or diamorphine<sup>6</sup> indicated that adequate analgesia could be achieved. However, motor blockade and operative deliveries occurred frequently, in keeping with other studies.<sup>2,3,7</sup> We have therefore undertaken this study to investigate whether further dilution of bupivacaine would reduce the incidence of these problems. We have used the same concentration of fentanyl in our three trial solutions and the same infusion regimen as in our previous study to allow direct comparison.

### Methods

The study was approved by the District Ethics Committee, and written informed consent was obtained from all participants. Healthy primiparous mothers with uncomplicated singleton pregnancies were included in the study, provided they had not received any systemic opioid and were expected to continue in labour for at least 2 hours. Women of less than 150 cm in height, more than 100 kg in weight or over 5 cm cervical dilation were not studied.

An intravenous cannula was inserted before performing the epidural, and the circulation preloaded with 1000 ml compound sodium lactate solution. A baseline pain score was obtained at the peak of a contraction using a 200 mm visual analogue scale. Approximately 3 cm of catheter was inserted at the second or third lumbar interspace with the mother in the lateral position. All patients then received a test dose of 0.5% bupivacaine 3 ml, followed by 0.5% bupivacaine 5 ml to establish analgesia, with further increments of 0.5% bupivacaine 3 ml given if necessary. If adequate analgesia was not obtained, the catheter was resited and the mother removed from the study.

Initial assessment of pain relief, motor blockade and sensory level was carried out by the anaesthetist, who then started the epidural infusion with one of three randomly allocated solutions. Group A received 0.125% bupivacaine,

H.A. Noble, BM, FCAnaes, Senior Registrar, G.R. Enever,\* MA, MB, BS, FCAnaes, Clinical Research Fellow, T.A. Thomas, MB, ChB, FCAnaes, Consultant, Department of Anaesthesia, Bristol Maternity Hospital, Southwell Street, Bristol BS2 8EG.

\*Present appointment: Senior Registrar, Royal Victoria Infirmary, Newcastle upon Tyne.

Accepted 24 September 1990.

group B 0.062% bupivacaine and group C 0.031% bupivacaine, all three with 0.0002% fentanyl. All infusions were run at 7.5 ml/hour. They were prepared by the anaesthetist performing the epidural using 30 ml, 15 ml or 7.5 ml of 0.25% bupivacaine and fentanyl 120 µg, diluted in a syringe to a final volume of 60 ml using 0.9% saline. The infusions were given via a bacterial filter using a syringe driver, and were continued until after delivery.

The mothers' posture was not controlled during the study, as long as the position adopted avoided aortocaval compression. Loss of temperature sensation to ethyl chloride spray was used to define the upper limit of sensory blockade; motor blockade was assessed using the Bromage scale.<sup>9</sup> These observations were repeated every hour after starting the study infusions. Arterial blood pressure, pulse rate, respiratory rate and fetal heart rate were recorded every 30 minutes. Specially trained midwives carried out all observations and were unaware of which solution the mother received.

If the mother complained of inadequate analgesia, additional top-up doses of 0.25% bupivacaine 8 ml were given.

Table 1. Demographic data on entering study; mean (SD).

	Group A (n = 21)	Group B (n = 17)	Group C (n = 18)
Age; years	24(5)	24(4)	28(5)
Height; cm	162(6)	162(7)	162(6)
Weight; kg	74(14)	79(11)	80(14)
Cervical dilation; cm	3.5(1.3)	3.6(1.3)	3.5(1.6)
Median pain score	148	185	155
(Range)	(78–200)	(90–200)	(89–200)

Table 2. Requirements for top-up injections of bupivacaine during infusion; number of patients, %; top-up interval and time to first top-up/delivery, minutes (SD). There were no significant differences between the three groups.

	Group A (n = 21)	Group B (n = 17)	Group C (n = 18)
No top-ups	10(48)	5(29)	6(33)
One top-up	5(24)	5(29)	4(22)
Two top-ups	3(14)	1(6)	3(17)
Three top-ups	1(5)	2(12)	2(11)
More than three top-ups	2(9)	4(24)	3(17)
Top-up interval	252(144)	190(118)	202(179)
First top-up/delivery	270(154)	215(112)	208(184)

The number of doses was recorded, as was the time to first top-up and the mean top-up interval<sup>10</sup> was calculated for each group.

The incidence of pruritus, nausea or vomiting was elicited by direct questioning during and after the period of infusion. Details of the mode of delivery and the condition of the infant were recorded. The mothers were interviewed the next day to assess any urinary problems, and the general quality and acceptability of the epidural analgesia they had received.

Statistical analyses were performed using the Chi-squared test, and analysis of variance (ANOVA) as appropriate.

## Results

Fifty-six primiparous women were included in the study, of whom 21 received 0.125% bupivacaine (group A), 17 0.062% bupivacaine (group B) and 18 0.031% bupivacaine (group C). The women in group C were older than the other groups, despite random allocation. There were no other statistical differences in the demographic data of the three groups (Table 1).

Requirements for top-up doses of 0.25% bupivacaine are shown in Table 2. There were no statistically significant differences between the three groups, although group A tended to require fewer than the other two groups. The times (SD) to either first top-up or delivery in the three groups were 270 (154) minutes (group A), 215 (112) minutes (group B) and 208 (184) minutes (group C). There was no statistical difference between the three groups.

There was no statistical difference in the mean top-up interval between the three groups, the values being 252 (144) minutes (group A), 190 (118) minutes (group B) and 202 (179) minutes (group C), or the mean duration of infusion, 427 (189) minutes (group A), 413 (159) minutes (group B) and 391 (193) minutes (group C).

The median visual-analogue pain scores (0–200) for each group are shown in Table 3. The median pain scores in group C were significantly higher for the first 4 hours of the study ( $p < 0.001$ ). This difference did not persist as the study progressed. Pain scores were also grouped under the following headings (Table 4); no pain, score of 0; mild pain, score of 1 to 60; moderate/severe pain, score of 61 to 200. Analysis of the pain scores in this manner showed no significant difference between the three groups.

Table 3. Median pain scores (range, 0–200) for the three groups during labour. n = number remaining undelivered in group.

Infusion time (hours)	Group A	n	Group B	n	Group C	n
1	1.5(0–86)	21	2(0–96)	17	26.5(0–115)	18
2	0(0–26)	21	0(0–105)	17	18.5(0–64)	18
3	0(0–50)	20	5(0–155)	17	30(0–150)	15
4	0(0–60)	17	2.5(0–100)	14	22(0–180)	10
5	46(0–115)	11	2(0–200)	13	19(0–190)	9
6	5(0–60)	9	100(0–200)	8	55(0–200)	7
7	12(0–71)	6	110(20–170)	6	92(0–200)	6
8	60(8–135)	5	10(0–107)	6	35(0–200)	4
9	60(24–106)	3	20(0–102)	5	110(0–160)	3
10	95(60–165)	3	4(0–126)	3	26(3–100)	3
11	55(30–80)	2	—	0	59(19–100)	2
12	101(65–138)	2	—	0	19	1

**Table 4.** Distribution of all pain scores for the three groups; number, %. There were no significant differences between the three groups.

Total pain scores	Group A	Group B	Group C
0	48(45)	45(43)	19(20)
1-60	51(47)	27(25)	51(53)
61-200	9(8)	34(32)	26(27)

The mean level of sensory blockade in all groups was T<sub>9</sub>, and the highest level recorded was T<sub>4</sub> in a woman in group C. Motor blockade was not statistically different between the three groups, although over half of the women in group B (53%) had no appreciable blockade, compared to 19% in group A and 33% in group C (Table 5). Urinary problems were difficult to assess, since it was common practice to catheterise patients before operative delivery. However, the incidence of problems after delivery was very low.

No symptoms associated with systemic toxicity of bupivacaine were noted during this study. The dose rate of fentanyl was 15 µg/hour and no evidence of respiratory depression, as judged by respiratory rate changes, was noted in any mother.

The incidence of other side effects are shown in Table 6. Only one woman in each group experienced pruritus. There was no significant difference in the condition of infants at one or 5 minutes, and no infants required naloxone or resuscitation other than normal bag and mask oxygen.

The mode of delivery is also shown in Table 6. There was no statistically significant difference between the three

**Table 5.** Incidence of motor blockade, using Bromage scale (4 = no blockade, 1 = full blockade); number of patients, %. There were no significant differences between the three groups.

Mean motor blockade (both legs)	Group A (n = 21)	Group B (n = 17)	Group C (n = 18)
Grade 4	4(19)	9(53)	6(33)
Grade 3 or above	12(57)	5(29)	6(33)
Less than grade 3 (either leg)	5(24)	3(18)	6(33)

**Table 6.** Incidence of side effects; number of patients, %, and median levels of sensory blockade. Mode of delivery; number of patients, %. There were no significant differences between the three groups.

	Group A (n = 21)	Group B (n = 17)	Group C (n = 18)
Decrease in systolic pressure > 20%	4(19)	3(18)	1(5)
Nausea only	0	2(12)	0
Nausea with vomiting	3(14)	3(18)	3(17)
Pruritus	1(5)	1(6)	1(6)
Apgar < 8 at 1 minute	4(19)	2(12)	4(22)
Apgar < 8 at 5 minutes	3(14)	0	1(6)
Sensory level	T <sub>9</sub>	T <sub>9</sub>	T <sub>9</sub>
Spontaneous vaginal	6(29)	10(59)	7(39)
Forceps	5(24)	4(24)	5(28)
Ventouse	6(29)	1(6)	2(11)
Caesarean section	4(19)	2(12)	4(22)

groups, although there were more spontaneous vaginal deliveries (59%) in group B (0.1 > p > 0.05).

## Discussion

### Analgesia

In this study we have demonstrated an increase in pain scores with increasing dilutions of bupivacaine. The scores were significantly higher ( $p < 0.001$ ) for the first 4 hours of infusion in the women in group C who received 0.031% bupivacaine. It is interesting that despite the relatively less effective analgesia in this group, the demand for top-up doses was not significantly different from the other two groups. Why this should happen is not obvious. It may be that some women will tolerate discomfort if they feel that they have some control over their pain should it deteriorate further. Solutions which appear less effective when assessed by pain scores may clinically be useful for women who prefer some sensation and involvement in their labours. The women in group B, who received 0.062% bupivacaine, had similar median pain scores to those receiving 0.125%. However, they had fewer pain scores of 0 and more of over 60 than the latter group.

### Neural blockade

We hoped to see a reduction in both motor and sensory blockade in the women who received more dilute bupivacaine, but this was not the case. This finding was in keeping with a previous study using 0.0625% bupivacaine.<sup>11</sup> We then further analysed our results to ascertain whether neural blockade was less in those women who had not received any top-ups, but again this was not the case.

### Delivery

We continued our infusions until delivery in an attempt to determine any benefit which dilute solutions might offer in terms of spontaneous deliveries. No significant difference was shown, however, and we believe this may be related to two factors. Firstly, the expected reduction in neural blockade did not occur. Secondly, a previous study<sup>12</sup> has suggested that adequate analgesia at delivery allows the mother to cooperate more fully and have the best chance of a normal delivery. The rate of normal, spontaneous vaginal delivery in this study was 39% (23 women) overall. Group B had a rate of 59% (10 out of 17 women) which is very good when compared to most previous studies.<sup>2,13</sup>

### Side effects

It was reassuring to demonstrate a similar low incidence of side effects to our previous study.<sup>6</sup> Pruritus only occurred in one woman in each of the three groups. Nausea with vomiting occurred equally in the three groups, in approximately one in five women. We detected no respiratory depression in this study by the limited and simple method of observing respiratory rate.

Our technique of using a low dose, low rate infusion with additional top-up doses of a different solution is, we believe, a fairly effective means of providing analgesia for labour. However, further benefit might be gained by a technique allowing variation in the base infusion rate, or by allowing the top-up to be of the infusion solution. This could be achieved using a single syringe patient-controlled analgesia type pump, controlled either by the midwife or the

patient.<sup>14</sup> However, this might reduce the safety of the technique by increasing the dose of opioid infused. Also, infusing large volumes of a dilute local anaesthetic has been shown to cause high neural blockade and a low normal delivery rate.<sup>15</sup>

Our choice of fentanyl for this study was made to allow us to compare this study with those of previous authors.<sup>3,13,16</sup> It may not be the best opioid for use in labour, as our recent study comparing it to diamorphine has indicated.<sup>6</sup>

This study has been unable to demonstrate any statistically significant advantages associated with the use of very dilute solutions for epidural pain relief during labour. We have demonstrated that 0.031% bupivacaine with 0.0002% fentanyl is inadequate for this purpose. However, further investigation of 0.062% bupivacaine is warranted, especially in combination with other opioids.

### References

1. EVANS KRL, CARRIE LES. Continuous epidural infusion of bupivacaine in labour: a simple method. *Anaesthesia* 1979; **34**: 310-5.
2. LAMONT RF, PINNEY D, ROGERS P, BRYANT TN. Continuous versus intermittent epidural analgesia: a randomised trial to observe obstetric outcome. *Anaesthesia* 1989; **44**: 893-6.
3. JONES G, PAUL DL, ELTON RA, MCCLURE JH. Comparison of bupivacaine and bupivacaine with fentanyl in continuous extradural analgesia during labour. *British Journal of Anaesthesia* 1989; **63**: 254-9.
4. REYNOLDS F. Extradural opioids in labour. *British Journal of Anaesthesia* 1989; **63**: 251-3.
5. COUSINS MJ, MATHER LE. Intrathecal and epidural administration of opioids. *Anesthesiology* 1984; **61**: 276-310.
6. ENEVER GR, NOBLE HA, KOLDITZ D, VALENTINE S, THOMAS TA. Epidural infusion of diamorphine with bupivacaine in labour: a comparison with fentanyl and bupivacaine. *Anaesthesia* (in press).
7. COHEN SE, TAN S, ALBRIGHT GA, HALPERN J. Epidural fentanyl/bupivacaine mixtures for obstetric analgesia. *Anesthesiology* 1987; **67**: 403-7.
8. THOMAS TA, GRIFFITHS MJ. A pain slide rule. *Anaesthesia* 1982; **37**: 960-1.
9. BROMAGE PR. *Epidural analgesia*. Philadelphia: W.B. Saunders, 1978.
10. BOGOD DG, ROSEN M, REES GAD. Extradural infusion of 0.125% bupivacaine at 10 ml h<sup>-1</sup> to women during labour. *British Journal of Anaesthesia* 1987; **59**: 325-30.
11. LI DF, REES GAD, ROSEN M. Continuous extradural infusion of 0.0625% or 0.125% bupivacaine for pain relief in primigravid labour. *British Journal of Anaesthesia* 1985; **57**: 264-70.
12. PHILLIPS KC, THOMAS TA. Second stage of labour with or without extradural analgesia. *Anaesthesia* 1983; **38**: 972-6.
13. WALTON P, REYNOLDS F. Epidural analgesia and instrumental delivery. *Anaesthesia* 1984; **39**: 218-23.
14. LYSAK SZ, EISENACH JC, DOBSON CE 2D. Patient-controlled epidural analgesia during labor: a comparison of three solutions with a continuous infusion control. *Anesthesiology* 1990; **72**: 44-9.
15. EWEN A, MCLEOD DD, MACLEOD DM, CAMPBELL A, TUNSTALL ME. Continuous infusion epidural analgesia in obstetrics: a comparison of 0.08% and 0.25% bupivacaine. *Anaesthesia* 1986; **41**: 143-7.
16. CHESTNUT DH, OWEN CL, BATES JN, OSTMAN LG, CHOI WW, GEIGER MW. Continuous infusion epidural analgesia during labor: a randomized, double-blind comparison of 0.0625% bupivacaine/0.0002% fentanyl versus 0.125% bupivacaine. *Anesthesiology* 1988; **68**: 754-9.

## Intra-operative patient-controlled sedation

### Comparison of patient-controlled propofol with anaesthetist-administered midazolam and fentanyl

G. A. OSBORNE, G. E. RUDKIN, N. J. CURTIS, D. VICKERS AND A. J. CRAKER

#### Summary

*The quality of sedation and postoperative recovery have been assessed for intra-operative sedation provided by either patient-controlled sedation with propofol or a standard method using divided doses of midazolam and fentanyl, in 40 ASA 1 day surgery patients undergoing extraction of third molar teeth under local analgesia. Patient-controlled sedation with propofol produced sedation no deeper than full eyelid closure with prompt response to verbal command, but deeper levels were seen in three patients in the midazolam and fentanyl group. Patient satisfaction was higher in the patient-controlled sedation propofol group for both subjective intra-operative feelings ( $p < 0.01$ ) and willingness to have the procedure again in the same manner ( $p < 0.05$ ). Amnesia was more limited to intra-operative events (rather than extending into the postoperative period) in the patient-controlled sedation propofol group ( $p < 0.05$ ). Drug dose was correlated with duration of procedure and surgical difficulty in the patient-controlled sedation propofol group but not in the midazolam and fentanyl group. Postoperative testing included a new computerised test, the FAST index, which indicated a dose-dependent reduction in cognitive function in the midazolam and fentanyl group, which persisted until the time of discharge. Changes in cognitive function in the patient-controlled sedation propofol group in the same postoperative interval were significantly less and not related to propofol dose.*

#### Key words

*Hypnotics, benzodiazepines; midazolam.  
Anaesthetic techniques; patient-controlled sedation.  
Anaesthesia; outpatient, recovery.  
Anaesthetics, intravenous; propofol.*

Patient-controlled sedation (PCS) with propofol has been shown to provide safe and effective intra-operative sedation for the extraction of third molar teeth under local analgesia.<sup>1</sup> In that earlier work, amnesia for significant postoperative events was noted to be absent, which is a significant advantage for day surgery patients. Our early clinical experience using PCS with propofol also suggested that postoperative recovery from drug effects was generally rapid and that this was likely to provide further advantages for day surgery.

We have undertaken this study to compare the quality of intra-operative sedation provided by PCS with propofol with that for a standard technique, using divided doses of midazolam and fentanyl, and to measure objectively postoperative recovery from drug effects with the two techniques. Psychometric testing used in the study to assess postoperative recovery includes the first anaesthesia-related application of a new computerised test of mental performance, the FAST (frequency accrual speed test) index.<sup>2</sup>

#### Patients and methods

This study was approved by the Hospital Human Ethics Committee. All operative procedures were carried out by one surgeon (N.C.). Forty fasted, unpremedicated, ASA 1 day surgery patients were randomly allocated to one of the two sedation regimens. All procedures were scored by the surgeon for surgical difficulty and patient cooperation, using analogue scales 0–10; these increased with surgical difficulty and patient cooperation. The obvious differences between the methods of sedation precluded the surgeon from being blinded to the sedation technique.

#### Group 1

Patients received intermittent doses of intravenous midazolam and fentanyl titrated to provide a comfortable patient with half to full eyelid closure and prompt response to verbal command. An arbitrary limit of a total dose of 100  $\mu$ g fentanyl was not exceeded, but no limit was placed on the total amount of midazolam.

G.A. Osborne, MB, BS, PhD, FFARACS, Staff Specialist, G.E. Rudkin, MB, BS, FFARACS, Senior Staff Specialist, Coordinator Day Surgery Unit, Department of Anaesthesia and Intensive Care, N.J. Curtis, BDS, FDS RCS, Registrar, Oral and Maxillofacial Surgical Unit, D. Vickers, BA, MA, PhD, Associate Professor, A.J. Craker, BSc, Research Assistant, Department of Psychology, University of Adelaide, Royal Adelaide Hospital, North Terrace, South Australia 5000.

Accepted 18 October 1990.

## Group 2

Patients, previously instructed in the use of the patient-controlled technique, self-administered 0.7 mg/kg intravenous bolus doses of propofol, delivered at 16.7 mg/minute, with a one-minute lockout interval. A modified, commercial, patient-controlled analgesia (PCA) pump<sup>1</sup> was used after an initial dose of 0.7 mg/kg propofol was given by the anaesthetist before the administration of local analgesia by the surgeon. All patients received oxygen by nasal cannula. Blood pressure, heart rate and haemoglobin oxygen saturation were monitored and recorded during all procedures.<sup>1</sup>

FAST index testing was carried out once before operation, then repeated during recovery when the patient first sat out of bed (no sooner than 30 minutes after arrival in the postoperative recovery room) and again at the time of discharge.

P-deletion<sup>3</sup> testing was carried out twice before operation (because of known practice effects) and twice after operation. The first postoperative test was at 10 minutes after arrival in the recovery room and the second at the time of discharge.

The time from arrival in recovery to first sitting out of bed and the time when ready for discharge were recorded. All patients were followed-up by telephone on the first postoperative day and asked a standard set of questions about peri-operative events. These included memory for intravenous cannulation, administration of local analgesia by the surgeon, extraction of teeth, recovery room events and journey home. Patients were also asked if they felt good, bad or indifferent during the procedure and if they would have the same procedure performed again in the same manner. Patients who used the PCS technique were asked if they liked the method of self-administration.

Data related to patient amnesia, intra-operative feelings and willingness to repeat the procedure in the same manner, were tested using the Fisher exact test (two-tailed). Pearson and Spearman correlation coefficients were calculated to determine the correlation between drug dose and procedure duration and surgical difficulty respectively. Repeated measures analysis of variance of the psychometric data was performed using program 5v, BMDP Statistical Software, UCLA, California, USA.

The FAST index requires the patient to make choices as to which of two adjacent rapidly flashing lights appears to flash more often.<sup>2</sup> Each test includes 100 trials and takes about 5 minutes to complete. We have used this new computerised test of mental speed for the first time as a

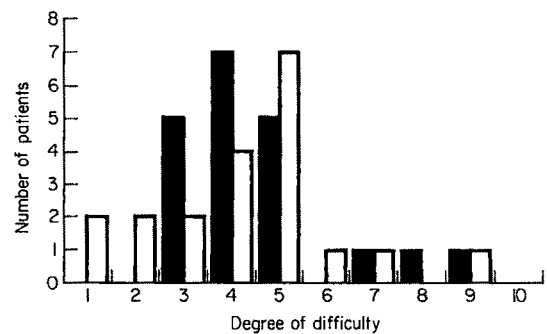


Fig. 1. Distribution of scores for surgical difficulty. ■, midazolam/fentanyl (group 1); □, PCS propofol (group 2).

measure of the effect of anaesthetic drugs on postoperative cognitive function. This test is convenient to use and is intended to provide a measure of mental speed that avoids some of the difficulties associated with choice reaction time (CRT)<sup>4</sup> and critical flicker frequency (CFF).<sup>5</sup> For example, CRT measurement may have learning components that can lead to underestimation of postoperative drug effects and may also be subject to speed-accuracy trade-off.

## Results

Summary data for patients and procedures are shown in Table 1. The two groups are well matched for weight, age, procedure duration, time to sitting in recovery and time when ready for discharge. Three patients in group 1 and one in group 2 were male. The distributions for surgical difficulty (Fig. 1) and patient cooperation (Fig. 2) are reasonably similar for both groups.

The average dose of propofol was 5.29 mg/kg (SD 2.68, range 2.10–12.04) while that of midazolam was 0.11 mg/kg (SD 0.04, range 0.05–0.16). The mean dose of fentanyl was 1.78 µg/kg (SD 0.28, range 1.00–2.30). Patients in the PCS group made an average of 6.7 (SD 3.8) successful demands and 31.6 (SD 53.6) unsuccessful during an effective lock-out period of about 3.5 minutes, as in addition to the 1 minute lockout, most self-administered boluses took about 2.5 minutes to deliver.

There was no evidence of cardiovascular or respiratory instability in either group. Haemoglobin oxygen saturation was maintained at 97% or greater for all patients. No patient in the PCS with propofol group was sedated to a level deeper than full eyelid closure with prompt response to verbal command. However, three patients in the midazolam and fentanyl group reached a deeper level where response required mild physical stimulus.

Table 1. Summary data for patients and procedures.

	Group 1 <i>n</i> = 20 Midazolam/ fentanyl		Group 2 <i>n</i> = 20 PCS propofol	
	Mean	SD	Mean	SD
Age; years	21.1	3.8	21.8	4.2
Weight; kg	59.9	12.6	56.3	6.8
Procedure time; minutes	42.7	17.1	47.5	15.7
Recovery time to sit; minutes	34.5	9.5	36.5	14.4
Recovery time to discharge; minutes	125.8	30.1	124.3	37.1

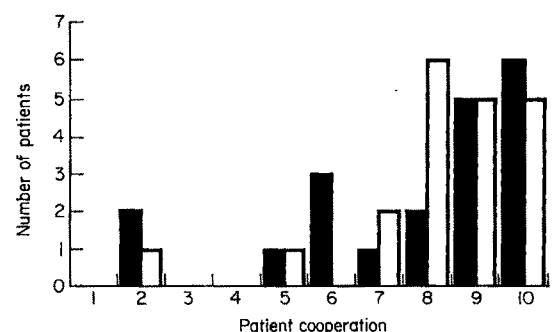


Fig. 2. Distribution of scores for patient cooperation. ■, midazolam/fentanyl (group 1); □, PCS propofol (group 2).

**Table 2.** Patient intra-operative subjective feelings.

	Group 1 <i>n</i> = 20 Midazolam/fentanyl	Group 2 <i>n</i> = 20 PCS propofol
Good	11	19
Indifferent	8	1
Bad	1	0

Patients in the group that received PCS with propofol were more satisfied with their sedation, as measured by patient subjective intra-operative feelings (Table 2) and patient willingness to have the same procedure again in the same manner (Table 3). More patients in the PCS propofol group stated they felt 'good' during the procedure ( $p < 0.01$ ) and more patients stated that they would have the procedure again in the same manner ( $p < 0.05$ ). All PCS patients said they enjoyed the experience of self-administration of sedation.

Peri-operative amnesia was more selective for intra-operative events in patients who received patient-controlled propofol (Table 4). While 30% of the midazolam and fentanyl group had amnesia for recovery room events, no patient who received PCS with propofol was amnesic for postoperative events ( $p < 0.05$ ).

Reasonable correlation existed between the mg/kg dose of self-administered propofol and both procedure duration ( $r = 0.441$ ;  $p = 0.052$ ) and surgical difficulty ( $r_s = 0.553$ ;  $p = 0.016$ ), but no corresponding correlation was seen with total midazolam dose ( $r = 0.099$ ;  $p = 0.678$  and  $r_s = -0.253$ ;  $p = 0.270$  respectively).

#### Results from psychometric testing

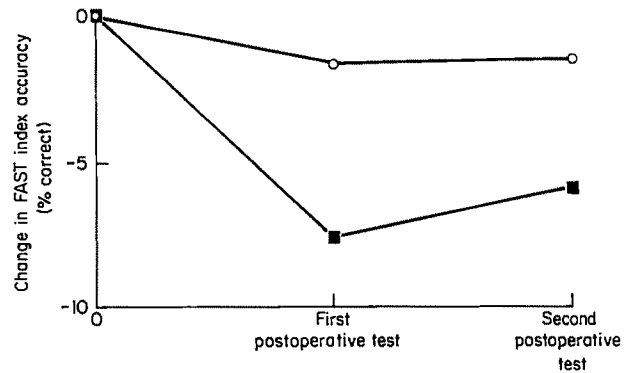
Grouped mean results for FAST index accuracy and number of ps correctly deleted are shown in Figures 3 and 4 respectively, for illustrative purposes only. Statistical analysis of these variables and the number of errors made in the p-deletion tests were analysed using repeated measures analysis of variance with an unstructured covariance matrix. Balancing of the two patient groups for

**Table 3.** Patient willingness to have procedure performed in the same manner.

	Group 1 <i>n</i> = 20 Midazolam/fentanyl	Group 2 <i>n</i> = 20 PCS propofol
Yes	15	20
Indifferent	4	0
No	1	0

**Table 4.** Patient amnesia for peri-operative events.

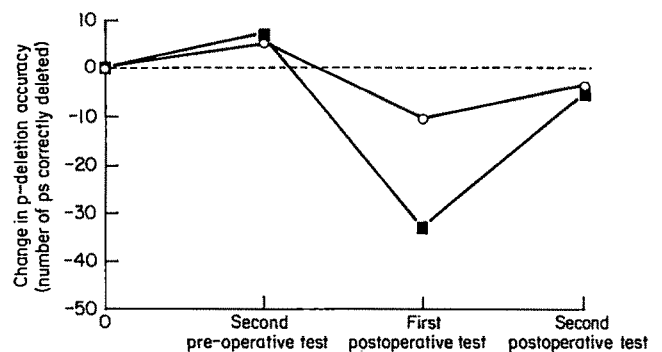
	Group 1 <i>n</i> = 20 Midazolam/ fentanyl	Group 2 <i>n</i> = 20 PCS propofol
Intravenous cannulation	1	0
Surgical local analgesia	4	4
Extractions	15	15
Postoperative recovery	6	0
Journey home	0	0

**Fig. 3.** Grouped mean results for FAST index accuracy. ■, midazolam/fentanyl (group 1); ○, PCS propofol (group 2).

various associated factors including surgeon, patient sex and age and surgical difficulty was attempted by experimental design and was found by marginal analysis to have been generally adequate. As a precaution, these factors were included in the formal analysis but no significant effect was detected for any of these factors for the three variables. Four covariates, predrug level of the variable, time after completion of the operative procedure when testing occurred, total midazolam dose (mg/kg) and total propofol dose (mg/kg) were also included in the analysis. These are variables that differ between individuals and cannot be easily balanced across treatment groups before sedation.

Specific findings were: *FAST index accuracy (f)*. (% correct). After excluding nonsignificant factors and covariates from the model: group 1 (midazolam and fentanyl). Mean  $f = 42.03 + 0.65$  (predrug  $f$ ) - 186.93 (midazolam dose (mg/kg)); group 2 (PCS propofol). Mean  $f = 28.61 + 0.65$  (predrug  $f$ ), i.e. the average FAST index accuracy of the PCS propofol group is approximately 6% units higher than the midazolam and fentanyl group, for any specified level of predrug  $f$ , with an average midazolam dose of about 0.1 mg/kg; this difference does not change significantly over the time interval between the two recovery room tests.

*Number of ps correctly deleted (p)*. After excluding nonsignificant factors and covariates from the model: group 1 (midazolam and fentanyl). Mean  $p = 0.225$  (recovery time (minutes)) + 0.876 (predrug  $p$ ) - 249.3 (midazolam dose (mg/kg)); group 2 (PCS propofol). Mean  $p = 0.093$  (recovery time (minutes)) + 0.876 (predrug  $p$ ), i.e. the average number of ps correctly deleted for the PCS propofol group is approximately 24 more than for the midazolam and fentanyl group, 10 minutes after arrival in recovery, for any

**Fig. 4.** Grouped mean results for number of ps correctly deleted. ■, midazolam/fentanyl (group 1); ○, PCS propofol (group 2).



specified level of predrug p, and average midazolam dose of around 0.1 mg/kg; this difference between the groups decreases with time over the recovery interval tested.

*Errors made in p-deletion.* There was no detectable difference between groups.

### Discussion

This study indicates that PCS with propofol can provide sedation with safety, effectiveness and patient satisfaction that compares favourably with a well known, standard technique. This is encouraging for the patient-controlled technique, since the surgical extraction of third molar teeth under local analgesia has higher requirements for sedation than many other procedures.

Advantages related to more selective amnesia and more rapid postoperative recovery from drug effects observed in the PCS group may be due more to the use of propofol than the patient-controlled technique. The relative contributions of the drug and the patient-controlled technique to any of the apparent advantages seen so far can be more precisely determined in further studies. It is possible that the effectiveness of the patient-controlled technique can be improved by further modification of patient-controlled pump parameters.

The psychometric testing in this study showed that patients who received PCS with propofol recovered more quickly from drug effects than patients who received the standard sedation technique with divided doses of midazolam and fentanyl. Midazolam had a dose dependent effect on both FAST index (cognitive function) and p-deletion (attention level) but there was no dose dependent effect for propofol for either test.

The FAST index results suggest that while there was little residual drug effect on cognitive function after 30 minutes in the postoperative recovery room in patients who received patient-controlled propofol, patients who received midazolam and fentanyl were significantly affected at 30 minutes postoperatively to an extent that persisted to the time of discharge, on average about 2 hours after operation. The effect of midazolam and fentanyl sedation on cognitive function, which persisted up to the time of discharge for day patients, suggests that patients so affected may have impaired performance in important everyday tasks, and further work is required to determine the duration and significance of this effect.

P-deletion, a well known test of attention level, was tested earlier in the recovery period when postoperative performance (number of ps correctly deleted) was reduced in both groups (midazolam and fentanyl more than PCS propofol). However, performance in both groups improved with time in recovery and approached pre-operative levels by the time of discharge; this suggests that the test may not be sensitive in its ability to discriminate between recovery from different drugs used for intra-operative sedation. The lack of drug effect on number of errors made (omitted ps and incorrectly deleted letters) is consistent with previous findings.<sup>6</sup>

The FAST index appears to be able to measure subtle decreases in cognitive ability. Work to date with the FAST index shows it provides a stable, valid and reliable measure of intellectual performance which is largely theory-neutral. In each of four studies, Vickers *et al.*<sup>7</sup> found that there was a reasonable correlation (averaging 0.45) between FAST

scores across two sessions, conducted one week apart, and that in none of these studies was there a significant difference in performance between the two sessions. In a later study with children, Vickers and McDowell<sup>2</sup> found that there was no significant difference between FAST scores in earlier and later sessions, but that there was good correlation ( $r = 0.69$ ,  $p < 0.01$ ,  $n = 28$ ) between FAST scores across the two sessions. These and other studies also provide evidence of validity of the FAST index as an index of mental speed. Vickers *et al.*<sup>7</sup> found a reasonable correlation ( $r = 0.52$ ,  $p < 0.01$ ,  $n = 22$ ) between FAST scores and intellectual ability on Raven's Advanced Progressive Matrices. In addition, Vickers and McDowell<sup>2</sup> found a high correlation between FAST scores and the Wechsler Intelligence scale for children ( $r = 0.69$ ,  $p < 0.01$ ,  $n = 28$ ). The ability of the FAST index, as demonstrated in this present study, to discriminate between recovery from different drugs and measure dose-dependent effects, further supports its validity as a measure of cognitive function.

The dose-dependent effect of midazolam on postoperative cognitive function gives us hope that more accurate mapping of drug dose and likely effect on cognitive function may be possible. This should be of particular value for day surgery patients. The ability to measure dose-dependent effects may mean that drug combinations designed to deliver optimal sedation and recovery characteristics can be explored with the FAST index.

### Acknowledgments

We are grateful for the valuable advice and assistance provided by P. I. Leppard, Department of Mathematical Statistics, University of Adelaide, who also carried out the repeated measures analysis of variance of the psychometric tests. We are also grateful for the cooperation and assistance provided by the nursing staff in the Day Surgery Unit and the Oral and Maxillofacial Unit of the Royal Adelaide Hospital. D. Vickers acknowledges support from an Australian Research Council grant (A 78615464).

### References

1. RUDKIN GE, OSBORNE GA, CURTIS NJ. Intraoperative patient-controlled sedation. *Anaesthesia*, (in press).
2. VICKERS D, McDOWELL A. Individual differences in the rate of accrual of information: the FAST (frequency accrual speed test) index. In: MULLER F, ed. *Proceedings of the Sixth Annual Meeting of the International Society for Psychophysics*, Würzburg. Würzburg: Institut für Psychologie. Universität Würzburg, 1990: 251-6.
3. DIXON RA, THORNTON JA. Tests of recovery from anaesthesia and sedation: intravenous diazepam in dentistry. *British Journal of Anaesthesia* 1973; **45**: 207-15.
4. SMITH EE. Choice reaction time: an analysis of the major theoretical positions. *Psychological Bulletin* 1968; **69**: 77-110.
5. SMITH JM, MISIAK H. Critical flicker frequency (CFF) and psychotropic drugs in normal human subjects—a review. *Psychopharmacology* 1976; **47**: 175-82.
6. ZUURMOND WWA, BALK VA, VAN DIS H, VAN LEEUWEN L, PAUL EAA. Multidimensionality of psychological recovery from anaesthesia: analysing six recovery tests. *Anaesthesia* 1989; **44**: 889-92.
7. VICKERS D, FOREMAN EA, NICHOLS MER, INNES NJ, GOTT RE. Some experimental tests of the application of an uncertainty model to a time-limited expanded judgement task. In: VICKERS D, SMITH PL, eds. *Human information processing: measure, mechanisms and models. Proceedings of the XXIV Congress of Psychology*, Vol 2. Amsterdam: Elsevier, 1989: 253-65.

CASE REPORT

## Fatal theophylline poisoning with rhabdomyolysis

### A potential role for dantrolene treatment

M. J. A. PARR AND S. M. WILLATTS

#### Summary

*A fatal case of theophylline poisoning is described. The patient developed rhabdomyolysis, renal failure and compartment syndrome, as well as the more usual features of severe theophylline poisoning. Dantrolene appeared to be useful in controlling the hypermetabolic state associated with the overdose and may have a role in future treatment.*

#### Key words

Poisoning; theophylline.

Neuromuscular relaxants; dantrolene.

#### Case history

A 22-year-old male asthmatic was admitted 2 hours after taking an intentional overdose of seventy 250 mg capsules of slow-release theophylline. On admission he was anxious and sweating with a heart rate of 70 beats/minute in sinus rhythm, blood pressure 80/40 mmHg, a respiratory rate of 20/minute and a Glasgow Coma Score of 15.

In the Accident and Emergency Department he received 30 ml of ipecacuanha which produced blood-stained vomit containing capsules. He was given activated charcoal 50 g orally, ranitidine 50 mg intravenously and was transferred to the Coronary Care Unit for electrocardiograph (ECG) monitoring.

Biochemical analysis showed a serum potassium concentration of 2.4 mmol/litre, glucose 12.2 mmol/litre and bicarbonate 17 mmol/litre; the serum theophylline concentration, reported initially as greater than 1000  $\mu$ mol/litre, was in fact 750  $\mu$ mol/litre (Table 1). The patient developed a supraventricular tachycardia with a rate of 140 beats/minute and received 500 ml gelatin solution, potassium chloride 40 mmol and amiodarone 300 mg intravenously. Blood gases whilst breathing air were  $P_{aCO_2}$  4.1 kPa,  $P_{aO_2}$  7.3 kPa and pH 7.31.

He developed ventricular tachycardia 4 hours after admission and was given disopyramide 100 mg intravenously with no effect. He then had a grand mal convulsion and was given diazepam 10 mg intravenously. He had a cardiac arrest in ventricular fibrillation. He was resuscitated after three 200 Joule DC shocks in addition to

adrenaline 3 mg and atropine 0.6 mg. His trachea was intubated, his lungs were ventilated and he was transferred to the Intensive Therapy Unit (ITU).

Therapy on ITU started with correction of the profound metabolic acidosis found after cardiac arrest (pH 6.55, bicarbonate 6 mmol/litre). Sodium bicarbonate (200 mmol) given in increments produced a pH of 7.28, a bicarbonate concentration of 18 mmol/litre and a  $P_{aCO_2}$  of 4.7 kPa. Aggressive potassium replacement was initiated; he received 100 mmol over the first 4 hours and 210 mmol over the next 8 hours, half as potassium phosphate ( $K_2HPO_4$ ) as the initial serum phosphate concentration was 0.51 mmol/litre. Activated charcoal (20 g 2-hourly) with lactulose (20 ml) was administered through a nasogastric tube and he continued to receive intravenous ranitidine.

Convulsions were refractory to infusion of midazolam but were controlled with a single dose of thiopentone 300 mg followed by an infusion of 375 mg/hour. Hyperglycaemia was controlled with an insulin infusion of up to 5 units/hour. Rapid atrial fibrillation (180 beats/minute) and hypotension (80/40 mmHg) were treated with metoprolol (10 mg) in increments which slowed the rate to 140 beats/minute and increased the arterial pressure to 90/50 mmHg, but the effect was short-lived. Practolol was ineffective.

His rectal temperature rose to 39.6°C and during spontaneous breathing through a ventilator (Servo 900C) with pressure support, he maintained a ventilatory minute volume in excess of 35 litres. Blood gas analysis revealed: pH 7.36;  $P_{aCO_2}$  2.8 kPa;  $P_{aO_2}$  35.2 kPa; and bicarbonate 11

M.J.A. Parr, MRCP, FCAnaes, Registrar, S.M. Willatts, FRCP, FCAnaes, Consultant, Sir Humphry Davy Department of Anaesthesia, Bristol Royal Infirmary, Bristol BS2 8HW.

Accepted 29 November 1990.

**Table 1.** Serum theophylline, creatinine, creatine kinase and potassium concentrations.

Hours after admission	Theophylline $\mu\text{mol/litre}$	Creatinine $\mu\text{mol/litre}$	Creatine kinase unit/litre	Potassium mmol/litre
0	750	124	108	2.4
4		183	550	3.6
10	1210	251	57 933	3.9
16	1078	281		3.7
20	1034	311	123 000	3.1
29	440	347	232 000	6.0

mmol/litre. The evidence of ongoing hypermetabolism led to treatment with dantrolene 1 mg/kg over 20 minutes then 2 mg/kg/hour for 4 hours; there were reductions in temperature and minute volume. His temperature was 38°C (decreasing to 37°C over the next 2 hours), the minute volume was 25 litres, heart rate was 140 beats/minute (atrial fibrillation) and arterial pressure 110/50 mmHg after treatment with dantrolene. An amiodarone infusion was started.

Renal function was deteriorating (plasma creatinine concentration 214  $\mu\text{mol/litre}$ ); urine with an osmolality of 351 mosmoles/kg contained large amounts of myoglobin. Serum creatine kinase concentration was 57 933 units/litre and theophylline concentration was 1210  $\mu\text{mol/litre}$ . Haemodiafiltration using femoral catheters was started and further renal support was provided with boluses of mannitol 25 g and a dopamine infusion of 2  $\mu\text{g/kg/minute}$ . Haemodiafiltration using 2 litre hourly dialysate exchange was used for 6 hours, but there was little change in serum theophylline concentration (1078  $\mu\text{mol/litre}$ ) although clinical stability had been achieved.

Twenty-four hours after ITU admission his heart rate was 80 beats/minute in sinus rhythm, arterial pressure 120/70 mmHg, ventilatory minute volume 16 litres, arterial pH 7.4,  $\text{Paco}_2$  4.3 kPa,  $\text{Pao}_2$  24.9 kPa and bicarbonate 22 mmol/litre. His temperature had remained normal and the serum theophylline concentration had decreased to 440  $\mu\text{mol/litre}$ . However, he had developed bilateral calf compartment syndromes, confirmed by pressure measurement, which required surgical decompression. The serum creatine kinase concentration had increased to over 232 000 units/litre and serum potassium concentration to 6.0 mmol/litre. He was treated by continued haemodiafiltration, a glucose and insulin infusion and enteral calcium resonium.

At the end of surgical decompression he had a hyperkalaemic cardiac arrest (serum potassium 9.0 mmol/litre) and died despite attempts at resuscitation. Postmortem examination revealed an intracerebellar haemorrhage, areas of hepatic necrosis, renal tubular necrosis with large myoglobin casts and gross changes of rhabdomyolysis in skeletal muscle with similar but less severe changes in cardiac muscle.

### Discussion

The mortality rate from theophylline poisoning has been estimated as 10%, and is greatly increased by the presence of severe manifestations e.g. convulsions, arrhythmias and cardiac arrest. The majority of deaths occur early, but 25% are delayed and result from complications.

The serum theophylline concentration of 1230  $\mu\text{mol/litre}$  represents massive absorption with a high probability of severe manifestations of toxicity and a poor outcome.<sup>2</sup> The relationship between serum concentration and toxicity is not straightforward; survival with no sequelae has been reported with higher serum concentrations than in our patient<sup>3,4</sup> but most reports of death or permanent neurological impairment are associated with much lower concentrations.

Our patient demonstrated many of the features of severe theophylline toxicity; anxiety, sweating, haematemesis, metabolic acidosis, hypokalaemia, hypophosphataemia, hyperglycaemia, leucocytosis, arrhythmias, hypotension, seizures and cardiac arrest occurred. His death resulted from hyperkalaemia secondary to rhabdomyolysis and renal failure which were apparent within 8 hours of admission.

Conventional treatment of theophylline poisoning relies on prevention of absorption, general supportive measures and augmenting elimination. Gastrointestinal absorption is reduced by emesis or lavage followed by enteral charcoal and the addition of a cathartic<sup>5,6</sup>. Supportive measures should include fluid, electrolyte, acid-base and glucose control. Treatment of seizures and arrhythmias can be difficult but while seizures may be refractory to benzodiazepines and phenytoin<sup>6</sup> there are no reports in which thiopentone was ineffective. Ventilatory and inotropic support may be necessary. The role of  $\beta$ -blockers in controlling arrhythmias is limited because of the risk of producing bronchospasm in asthmatics, but benefits have been reported<sup>7</sup> and attributed to the nonselective nature of propranolol. The cardioselective drug metoprolol produced short-lived improvement in our patient but was found to be of no value in a previous report.<sup>8</sup>

Exchange transfusion and peritoneal dialysis are inefficient methods of enhancing elimination of theophylline.<sup>6</sup> Enteral activated charcoal enhances elimination as well as preventing absorption, is cheap and easily administered. Charcoal haemoperfusion is the most efficient means of increasing elimination but is not without morbidity and, as in our case, is not always available. There has been no controlled study to compare outcome after haemoperfusion rather than supportive therapy alone, although a benefit from haemoperfusion has been suggested from retrospective evidence.<sup>9</sup>

Supportive therapy without haemoperfusion has been associated with excellent outcome even in very severe cases.<sup>3,4,10</sup> Haemodialysis has been used to increase clearance but is less efficient than charcoal haemoperfusion; in our case, the lack of improvement in serum theophylline concentration probably reflects continued gastrointestinal absorption despite charcoal administration.

Proposed mechanisms of action of the methylxanthines include augmenting translocation of intracellular calcium, increasing cyclic AMP concentration by phosphodiesterase inhibition and adenosine receptor blockade. Some recent evidence suggests that theophylline-enhanced thermogenesis in rats may be due at least in part to antagonism of adenosine receptors.<sup>11</sup> Caffeine, another methylxanthine, is a known trigger agent for *in vitro* testing of malignant hyperpyrexia susceptibility, the signs of which are similar to those shown by our patient. Dantrolene, which may uncouple the excitation-contraction of skeletal muscle by interfering with calcium release from the sarcoplasmic reti-

culum, may be as useful in the hyperpyrexia associated with the hypermetabolic state of severe theophylline poisoning as it is in the treatment of malignant hyperthermia. The clinical condition of our patient improved with dantrolene but the serum creatine kinase concentration continued to increase, reaching 232 000 units/litre.

Theophylline poisoning as a cause of rhabdomyolysis seems to be uncommon or under-reported and convulsions, hypoxia, hypotension, hypokalaemia and other drug ingestion may contribute to it. There are few case reports<sup>8,12-15</sup> and in a review of 399 cases of theophylline poisoning, rhabdomyolysis is mentioned as occurring in only three patients.<sup>1</sup> The small number of patients who develop rhabdomyolysis may share some susceptibility to the effects of theophylline which may be myotoxic in some circumstances. Theophylline has been implicated as a cause of myocarditis.<sup>16</sup> The cause of death in our patient was hyperkalaemia, possibly worsened by the fasciotomy<sup>17</sup> but clearly related to the profound muscle damage. Dantrolene controlled the hypermetabolism and might have resulted in a better outcome if given earlier. Further study of dantrolene in theophylline poisoning seems justified.

### References

1. PALOUEK FP, RODVOLD KA. Evaluation of theophylline overdoses and toxicities. *Annals of Emergency Medicine* 1988; **17**: 135-44.
2. ZWILLICH CW, SUTTON FD, NEFF TA, COHN WM, MATTHAY RA, WEINBERGER MM. Theophylline-induced seizures in adults. Correlation with serum concentrations. *Annals of Internal Medicine* 1975; **82**: 784-7.
3. DEAN LS, BROWN JW. Massive theophylline overdose. Survival without hemoperfusion. *Journal of the American Medical Association* 1982; **248**: 1742.
4. WELLS DH, FERLAUTO JJ. Survival after massive aminophylline overdose in a premature infant. *Pediatrics* 1979; **64**: 252-3.
5. GOLDBERG MJ, SPECTOR R, PARK GD, JOHNSON GF, ROBERTS P. The effect of sorbitol and activated charcoal on serum theophylline concentrations after slow-release theophylline. *Clinical Pharmacology and Therapeutics* 1987; **41**: 108-11.
6. GAUDREAU P, GUAY J. Theophylline poisoning. Pharmacological considerations and clinical management. *Medical Toxicology* 1986; **1**: 169-91.
7. BIBERSTEIN MP, ZIEGLER MG, WARD DM. Use of beta-blockade and hemoperfusion for acute theophylline poisoning. *Western Journal of Medicine* 1984; **141**: 485-90.
8. WHYTE KF, ADDIS GJ. Toxicity of salbutamol and theophylline together. *Lancet* 1983; **2**: 618-9.
9. WOO OF, POND SM, BENOWITZ NL, OLSON KR. Benefit of hemoperfusion in acute theophylline intoxication. *Journal of Toxicology, Clinical Toxicology* 1984; **22**: 411-24.
10. PARR MJA, DAY AC, KLETCHKO SL, CRONE PD, RANKIN APN. Theophylline poisoning. A review of 64 cases. *Intensive Care Medicine* 1990; **16**: 394-8.
11. WANG LCH, JOURDAN ML, LEE TF. Mechanisms underlying the supra-maximal thermogenesis elicited by aminophylline in rats. *Life Sciences* 1989; **44**: 927-34.
12. MACDONALD JB, JONES HM, COWAN RA. Rhabdomyolysis and acute renal failure after theophylline overdose. *Lancet* 1985; **1**: 932-33.
13. RUMPF KW, WAGNER H, CRIE CP, SCHWARCK H, KLEIN H, KREUZER H, SCHELER F. Rhabdomyolysis after theophylline overdose. *Lancet* 1985; **1**: 1451-2.
14. ESHLEMAN SHS, SHAW LM. Massive theophylline overdose with atypical metabolic abnormalities. *Clinical Chemistry* 1990; **36**: 398-9.
15. LLOYD DM, PAYNE SPK, TOMSON CRV, BARNES MR, ALLEN MJ. Acute compartment syndrome secondary to theophylline overdose. *Lancet* 1990; **336**: 312.
16. Myocarditis related to drug hypersensitivity. *Lancet* 1985; **2**: 1165-6.
17. HAIMOVICI H. Muscular, renal and metabolic complications of acute arterial occlusions: myoneuropathic-metabolic syndrome. *Surgery* 1979; **85**: 461-8.

## Epidural analgesia in Eaton–Lambert myasthenic syndrome

### Effects on respiratory function

S. SAKURA, Y. SAITO, M. MAEDA AND Y. KOSAKA

#### Summary

*The anaesthetic management of a patient with Eaton–Lambert myasthenic syndrome undergoing thoracotomy is described. Epidural anaesthesia, in combination with a light level of general anaesthesia, provided good operating conditions and postoperative analgesia. Pulmonary function and ventilatory responses to carbon dioxide and hypoxia were measured before operation. These tests were repeated after the epidural administration of 8 ml 2% lignocaine before surgery and after 2 mg morphine sulphate in 10 ml saline postoperatively. Minor reductions in some of the ventilatory parameters were observed. The epidural technique appears to be a useful and safe method by which to manage patients with Eaton–Lambert syndrome undergoing thoracotomy.*

#### Key words

*Complications; Eaton–Lambert (Lambert–Eaton) myasthenic syndrome. Anaesthetic techniques, regional; epidural.*

It is well known that patients with neuromuscular disorders may show abnormal sensitivity to neuromuscular blocking agents and that the administration of these drugs can cause difficulties during anaesthesia or in the postoperative period. Such patients might, therefore, be suitable candidates for epidural analgesia. The use of this technique has been reported in myasthenia gravis<sup>1</sup> and amyotrophic lateral sclerosis.<sup>2</sup> There has not been widespread acceptance of the use of epidural analgesia in neuromuscular disorders, however, because of the theoretical possibility that it may cause impairment of respiratory function.

A patient with Eaton–Lambert myasthenic syndrome (ELMS) and suspected carcinoma of the lung presented for thoracotomy. We were unable to find any reference to epidural analgesia in this syndrome. We decided, therefore, to use epidural analgesia as part of the anaesthetic technique and for pain relief after operation. The effects of epidural lignocaine and epidural morphine on the patient's pulmonary function were studied.

#### Case report

A 57-year-old man with ELMS, diagnosed by characteristic electromyographic (EMG) findings, was to undergo exploratory thoracotomy and an excision biopsy for suspected carcinoma of the lung. He had noticed the onset of weakness and aching in his extremities approximately 8

months earlier. He had had no remarkable past medical history.

Pre-operative evaluation revealed a middle-aged man, 57 kg in weight and 159 cm in height, with weakness of the upper and lower extremities. He had difficulty in climbing stairs and rising from a chair. His arterial blood pressure was 138/81 mmHg, pulse 61 beats/minute, and respiration 15 breaths/minute. No abnormalities were found on examination of the heart and lungs. He had no antibodies to acetylcholine receptors, a negative Tensilon (edrophonium) test and classical neurological findings of reduced deep tendon reflexes and no sensory changes. Chest X ray and computerised axial tomography showed a 3-cm mass in the right upper lobe with bilateral hilar lymph node enlargement.

The patient gave written informed consent before surgery to participate in data collection protocols approved by the Ethics Committee of Shimane Medical University. The following measurements were performed the day before surgery: pulmonary function tests, arterial blood gas analysis while breathing room air, and ventilatory responses to carbon dioxide and hypoxia. Pulmonary function was assessed using a Gould–Godart spirometer and computer, with the patient in a supine position. Functional residual capacity was measured by the closed-circuit helium dilution technique. Ventilatory sensitivities to CO<sub>2</sub> and hypoxia were determined using a modified Read re-

S. Sakura, MD, Y. Saito, MD, Consultants, M. Maeda, MD, Registrar, Y. Kosaka, MD, PhD, Professor and Chairman, Department of Anesthesiology, Shimane Medical University, 89-1 Enya-cho, Izumo 693, Japan.  
Accepted 15 November 1990.

**Table 1.** Lung volumes and dynamic tests of ventilatory capacity one day before surgery and 20 minutes after the epidural injection of lignocaine.

	Before epidural	After epidural
FVC; litres	2.82	2.39
FEV <sub>1.0</sub> ; litres	2.32	1.94
PEF; litres/second	6.83	5.33
FEF <sub>25-75%</sub> ; litres/second	2.52	2.28
FRC; litres	1.46	1.37
IC; litres	2.74	2.18
ERV; litres	0.31	0.33
RV; litres	1.11	1.02
TLC; litres	4.17	3.55
RV/TLC; %	26.6	28.7

FEV<sub>1.0</sub> = forced expired volume in 1 second; PEF = peak expiratory flow; FEF<sub>25-75%</sub> = forced expiratory flow over the mid-portion of vital capacity that is, from 25 to 75% of expired volume.

breathing method<sup>3</sup> and a modified Rebeck rebreathing method,<sup>4</sup> respectively. The results are summarised in Tables 1 and 2. After the measurements, with the patient in a lateral decubitus position, an epidural catheter (Portex) was introduced into the C<sub>7</sub>T<sub>1</sub> intervertebral space using the hanging drop technique with a 17-gauge Tuohy needle. A test dose of 2% lignocaine 2 ml was injected into the epidural catheter without evidence of intravascular injection or subarachnoid block.

On the day of surgery, the patient was premedicated with hydroxyzine 50 mg and atropine 0.5 mg intramuscularly 1 hour before admittance into an operating room, where 16-gauge intravenous and 22-gauge radial artery catheters were inserted. Twenty minutes after the epidural injection of 2% lignocaine 8 ml, pulmonary and ventilatory evaluation was again performed, as described above. The patient was then tested for sensation to pinprick which showed the distribution of analgesia to be from C<sub>5</sub>-T<sub>7</sub> bilaterally. The plasma concentration of lignocaine was 2.1 µg/ml. The vital capacity, inspiratory capacity and forced expiratory volume in 1 second decreased (Table 1). The Pao<sub>2</sub> and the mean slope of the CO<sub>2</sub> response curve increased, while that of the hypoxic response curve slightly decreased (Table 2).

Anaesthesia was induced with thiamylal 100 mg and the trachea was intubated following inhalation of nitrous oxide, oxygen and halothane. The patient was maintained with a light level of general anaesthesia intra-operatively; this consisted of 50% nitrous oxide in oxygen and 0.3% halothane. Three intermittent injections of 2% lignocaine 6 ml were given and artificial ventilation of the lungs was maintained without the use of neuromuscular blockers. The intra-operative course was uneventful. The patient's blood pressure remained within 10% of his pre-anaesthetic level, apart from a transient decrease immediately after induction of anaesthesia, with a pulse rate of approximately 75 beats/minute. The total operating time was 2 hours. On completion of surgery, morphine sulphate (2 mg in 10 ml saline) was injected through the epidural catheter for post-operative pain relief. The patient rapidly recovered from anaesthesia, his tracheal tube was removed and he was taken to an intensive care unit (ICU).

The postoperative course in the ICU was also uncomplicated and in particular there were no pulmonary complications. Epidural morphine successfully controlled the patient's pain without any need for additional analgesia,

**Table 2.** Arterial blood-gas tensions while breathing room air, resting ventilation, and ventilatory response to CO<sub>2</sub> and hypoxia one day before surgery, 20 minutes after the epidural injection of lignocaine, and 3 hours after morphine injection.

	Before epidural	Epidural lignocaine	Epidural morphine
Pao <sub>2</sub> ; kPa	9.23	11.04	9.08
Paco <sub>2</sub> ; kPa	5.67	5.36	5.08
Resting f; breaths/minute	13	13	13
Resting VT; ml	550	479	556
Resting VE; litres/minute	6.8	6.4	7.2
CO <sub>2</sub> response			
ΔVE/ΔPE'CO <sub>2</sub> ; litres/minutes kPa	24.7	32.9	30.1
VE <sup>7.3</sup> ; litres/minute	13.09	16.47	16.18
Hypoxic response			
ΔVE/ΔSao <sub>2</sub> ; litres/minute % O <sub>2</sub> Sat	2.36	1.83	1.62
VE <sup>95</sup> ; litres/minute	17.19	17.96	15.08

f = respiratory frequency; VT = tidal volume; VE = minute ventilation; PE'CO<sub>2</sub> = end-tidal CO<sub>2</sub> tension; VE<sup>7.3</sup> = minute ventilation at Pao<sub>2</sub> of 7.3 kPa; VE<sup>95</sup> = minute ventilation at Sao<sub>2</sub> of 95%.

and without producing respiratory depression. This was confirmed, not only by the frequent evaluation of respiratory frequency and arterial blood gas tensions, but also by repeated ventilatory measurements at 3 hours after the epidural injection of morphine (Table 2). At this time the plasma concentration of lignocaine was 2.4 µg/ml and that of morphine 2.8 ng/ml. On the second postoperative day, the patient was transferred to the open ward and his recovery was uneventful. Pathological examination of the tissue removed revealed a small cell carcinoma.

### Discussion

ELMS is a rare disorder of neuromuscular transmission characterised by progressive weakness of limb-girdle muscles and fatigability.<sup>5</sup> As the disorder is frequently associated with small cell carcinoma of the lung, many patients with ELMS require anaesthesia for biopsy and/or treatment of their primary malignancy. Anaesthetic considerations in ELMS include an exaggerated response to both depolarising and nondepolarising neuromuscular blocking drugs and an increased risk of postoperative respiratory failure that requires prolonged ventilatory assistance. In addition, recent reports have suggested the involvement of respiratory muscles and the diaphragm in ELMS,<sup>6</sup> and the possibility of the spontaneous development of respiratory failure.<sup>7</sup> Thus the use of neuromuscular blockers should be avoided if possible and inhalational anaesthetics alone have been advocated for these patients.<sup>5</sup> The possibility exists, however, that adequate muscle relaxation cannot be provided solely by inhalational agents.

The case described here demonstrated that epidural analgesia, which provides excellent surgical conditions when combined with a light level of general anaesthesia, could be an alternative to the use of neuromuscular blockers. Furthermore, its efficacy in providing postoperative pain relief was an additional advantage.

The respiratory measurements in this case were designed to determine whether or not epidural analgesia and post-

operative epidural morphine were safe for an ELMS patient from the aspect of pulmonary and ventilatory function. Our patient had both the typical clinical presentation and the results of EMG study to confirm the diagnosis of ELMS. Although we did not compare this patient with one without ELMS, this technique appeared to have little deleterious effect on lung function as judged by the results shown in the tables. In general, high levels of epidural anaesthesia are associated with a decrease in some lung and expired gas volumes.<sup>8</sup> The slight decrease in these volumes seen in our patient is, therefore, not specific to ELMS. Furthermore, the results of the hypercapnic and hypoxic ventilatory response tests suggested that epidural analgesia did not give rise to ventilatory depression. On the other hand, epidural morphine usually induces respiratory depression as a result of the rostral spread of morphine in the cerebrospinal fluid (CSF).<sup>9</sup> Although our single measurement of ventilatory response performed 3 hours after the epidural injection of morphine only demonstrated a lack of severe respiratory depression in the early phase, as far as we could judge from our routine respiratory monitoring late respiratory depression did not occur either. However, since the movement of the cervical epidural morphine to the cervical CSF would be expected to deliver more morphine to the brainstem than a more caudal approach, insertion of the epidural catheter rather lower in the thoracic region should perhaps be recommended.

In summary, the use of epidural analgesia combined with

light general anaesthesia and postoperative epidural morphine appears to be a safe technique with which to manage patients with ELMS undergoing thoracotomy.

### References

1. BURGESS FW, WILCOSKY B. Thoracic epidural anaesthesia for transsternal thymectomy in myasthenia gravis. *Anesthesia and Analgesia* 1989; **69**: 529-31.
2. KOCHI T, OKA T, MIZUGUCHI T. Epidural anesthesia for patients with amyotrophic lateral sclerosis. *Anesthesia and Analgesia* 1989; **68**: 410-12.
3. READ DJC. A clinical method for assessing the ventilatory response to carbon dioxide. *Australasian Annals of Medicine* 1967; **16**: 20-32.
4. REBUCK AS, CAMPBELL EJM. A clinical method for assessing ventilatory response to hypoxia. *American Review of Respiratory Disease* 1974; **109**: 345-50.
5. MILLER J, LEE C. Muscle diseases. In: KATZ J, eds. *Anesthesia and uncommon diseases: pathophysiologic and clinical correlations*, 2nd edn. Philadelphia: W.B. Saunders, 1981; 530-61.
6. WILCOX PG, MORRISON NJ, ANZARUT AR, PARDY RL. Lambert-Eaton myasthenic syndrome involving the diaphragm. *Chest* 1988; **93**: 604-6.
7. GRACEY DR, SOUTHERN PA. Respiratory failure in Lambert-Eaton myasthenic syndrome. *Chest* 1987; **91**: 716-8.
8. TAKASAKI M, TAKAHASHI T. Respiratory function during cervical and thoracic extradural analgesia in patients with normal lungs. *British Journal of Anaesthesia* 1980; **52**: 1271-76.
9. BROMAGE PR, CAMPORESI EM, DURANT PAC, NIELSEN CH. Rostral spread of epidural morphine. *Anesthesiology* 1982; **56**: 431-6.



## Chemical meningism after lumbar facet joint block with local anaesthetic and steroids

S. J. THOMSON, D. M. LOMAX AND B.-J. COLLETT

### Summary

*A case is reported in which chemical meningism occurred after lumbar facet joint block with methylprednisolone acetate and bupivacaine. This complication was probably due to inadvertent dural puncture. The use of steroids in facet joint injections is questioned.*

### Key words

*Pain; chronic.*

*Complications; chemical meningitis.*

*Anatomy; lumbar facet joint.*

*Hormones; corticosteroids.*

### Case history

A 44-year-old woman presented to the Pain Management Clinic with a 20-year history of low back and right buttock pain with occasional radiation to the right knee. She had first injured her back as a midwife whilst assisting at a delivery. Conservative treatment for the pain was not successful and she developed progressive numbness of her right leg. A laminectomy and removal of L<sub>4-5</sub> and L<sub>5</sub> S<sub>1</sub> discs was performed. The numbness in her leg gradually resolved but she continued to have intermittent episodes of severe back pain. On examination she was found to have reduced straight leg raising of 40° on the right side but no other abnormal neurological signs were found. Computerised axial tomography (CT) and magnetic resonance imaging (MRI) showed degenerative disc disease at L<sub>4-5</sub>, with a posterior bulge of the annulus without root compression. Facetal joint hypertrophy was also shown.

A series of diagnostic blocks with bupivacaine and methylprednisolone acetate were performed, which produced pain relief for 6 weeks, but by 4 months her symptoms had returned. Facet joint blocks were repeated on the right at L<sub>3-4</sub> and L<sub>4-5</sub>, together with a block of the medial branch of the posterior ramus of L<sub>3</sub>. A total of

12 ml 0.5% bupivacaine and 80 mg methylprednisolone acetate was used. The patient complained of severe pain in the leg during the injection of the posterior ramus. The injection was discontinued and the needle withdrawn, with resolution of the pain. Fifteen minutes after the procedure she complained of a mild parietal headache and a burning sensation throughout her body. She was flushed and had a tachycardia but remained normotensive. She had a temperature of 37.5°C. Bed rest, fanning and aspirin therapy was recommended overnight. The next day she had occipital headache, mild photophobia, marked neck stiffness and a temperature of 38.5°C. Neurological examination was otherwise normal. A full blood count, biochemistry, blood and urine cultures and chest X ray were negative. Chemical meningism was diagnosed but the decision was made not to perform a lumbar puncture unless there was evidence of neurological deterioration. The patient was given intravenous fluid therapy and codeine phosphate was prescribed for her headache. She developed a sensorineural hearing deficit in her right ear which lasted 2 days. She complained of blurred vision and a nonspecific skin pruritus. A CT head scan at this time was normal. Her occipital headache, photophobia, pyrexia

S.J. Thomson, MB, BS, FFARCS, Senior Registrar in Anaesthetics and Pain Management, D.M. Lomax, MB, BS, Registrar in Anaesthetics, \*B.-J. Collett, MB, BS, FFARCS, Consultant in Anaesthetics and Pain Management, Whipps Cross Hospital, Leytonstone, London E11 1NR.

Accepted 29 November 1990.

\*Present address: Leicester Royal Infirmary, Infirmary Square, Leicester LE1 5WW.

and neck stiffness completely resolved over 12 days and she was discharged from hospital.

At follow-up one month later she had a mild visual disturbance and persistent pruritus. Her back pain was considerably improved but she was anxious to know her future treatment should the pain recur.

### Discussion

Extradural injections of steroids and local anaesthetics have been used in the treatment of back pain and sciatica for over 30 years.<sup>1,2</sup> More recently, lumbar facet joint disease has been implicated as a cause of low back pain and it has been suggested that injection of this joint, or its nerve supply, with local anaesthetic and steroid will produce prolonged pain relief.<sup>3</sup>

However, there is still doubt about the efficacy of such treatments, or, if they are effective, the mechanism of action. In addition, the use of steroids in the proximity of the spinal cord and nerve roots has been questioned, as it is known that both soluble and depot preparations can have toxic neurological effects when injected into the subarachnoid space.<sup>4</sup> All preparations of steroids contain vehicles or preservatives that are neurotoxic. Methylprednisolone acetate is produced in a suspension of polyethylene glycol, myristyl-gamma-picolinium chloride and saline. Each millilitre of methylprednisolone contains an average of 30 mg polyethylene glycol, which is an alcohol and a nonionic detergent.<sup>4</sup> Application of polyethylene glycol in a 1:8 dilution to nerve, muscles and connective tissue has been shown to cause tissue necrosis.<sup>5</sup>

It would appear that extradural steroids are safer than those given by the subarachnoid route. In cats, single extradural injections of triamcinolone acetate produced only mild histological changes in the spinal root and meninges.<sup>6</sup> However, even the extradural route is not entirely free of complications and cases of extradural abscess and chemical meningitis have been reported.<sup>7,8</sup> It is possible that neurological complications associated with extradural steroids are a result of leakage of steroid into the subarachnoid space. This could occur either by inadvertent dural puncture or via some of the arachnoid villi which have been shown to penetrate the root dura.<sup>9</sup> Even in skilled hands, the incidence of inadvertent dural puncture during extradural analgesia is 0.5–2.5%.<sup>10</sup> In the case of chemical meningitis following extradural injection of methylprednisolone reported by Gutknecht, accidental dural puncture was thought to have been the cause.<sup>11</sup>

It is also possible to puncture the dural cuff during injection of the lumbar facet joint, although no case of chemical meningism has previously been reported in association with this block. In our patient, subarachnoid injection must have occurred during the attempt to block

the medial branch of the posterior ramus of L<sub>3</sub>. Direct introduction of the steroid into the subarachnoid space is suggested by the appearance of the first symptoms within 15 minutes of the injection. In Gutknecht's case, symptoms of chemical meningism did not develop until 4 hours later.

It is not known how or why analgesia is produced when steroids are injected into the facet joints. It has been suggested that the analgesia is not secondary to an anti-inflammatory action of the steroids, but due to a neurotoxic effect of polyethylene glycol denervating the small capsular nerve endings.<sup>12</sup>

In a recent review article on the treatment of backache, Kepes and Duncalf concluded that the rationale for the use of methylprednisolone, given by the extradural or intrathecal route, was not scientifically proven.<sup>13</sup> There is still no definitive controlled study of the efficacy of intra-articular steroids in the treatment of backache due to the facet syndrome and such a study would be welcomed.

### References

- BOUDIN G, BARBIZET J, GUIMARD J. L'hydrocortisone intra-arachnoidienne, ses applications cliniques en particulier dans le traitement de la méningite tuberculeuse. *Bulletin de la Société Médicale des Hôpitaux de Paris* 1955; **21**: 807–21.
- LIEVRE JA, BLOCH-MICHEL H, ATTALI P. L'injection trans-sacrée: clinique et radiologique. *Bulletin de la Société Médicale des Hôpitaux de Paris* 1957; **73**: 1110–7.
- MOONEY V, ROBERTSON J. The facet syndrome. *Clinical Orthopaedics and Related Research* 1976; **115**: 149–56.
- NELSON DA. Dangers from methylprednisolone acetate therapy by intraspinal injection. *Archives of Neurology* 1988; **45**: 804–6.
- MARGOLIS G, HALL HE, NOWILL WK. An investigation of efocaine, long-acting local anesthetic agent; animal studies. *Archives of Surgery* 1953; **67**: 715–30.
- DELANEY TJ, ROWLINGSON JC, CARRON H, BUTLER A. Epidural steroid effects on nerve and meninges. *Anesthesia and Analgesia* 1980; **59**: 610–4.
- GOUCKE CR, GRAZIOTTI P. Extradural abscess following local anaesthetic and steroid injection for chronic low back pain. *British Journal of Anaesthesia* 1990; **65**: 427–9.
- SHEALY CN. Dangers of spinal injection without proper diagnosis. *Journal of the American Medical Association* 1966; **197**: 1104–6.
- SHANTHA TR, EVANS JA. The relationship of epidural anesthesia to neural membranes and arachnoid villi. *Anesthesiology* 1972; **37**: 543–57.
- ALBRIGHT GA. Lumbar epidural anesthesia. In: *Anesthesia in obstetrics: maternal, fetal and neonatal aspects*. Menlo Park, Calif: Addison Publishing Co, 1978: 224–46.
- GUTKNECHT DR. Chemical meningitis following epidural injections of corticosteroids. *American Journal of Medicine* 1987; **82**: 570.
- BOGDUK N. Back pain: zygapophysial blocks and epidural steroids. In COUSINS MJ, BRIDENBAUGH PO, eds. *Neural blockade*, 2nd edn. Lippincott, 1988: 935–54.
- KEPES ER, DUNCALF D. Treatment of backache with spinal injections of local anesthetics, spinal and systemic steroids. A review. *Pain* 1985; **22**: 33–47.

## Tracheal tube resistance and airway and alveolar pressures during mechanical ventilation in the neonate

T. R. TIPPING AND M. K. SYKES

### Summary

The relationship between peak airway pressure, alveolar pressure and respiratory frequency was calculated for the range of compliances and airway resistances which might be encountered during mechanical ventilation of a 3-kg neonate. The pressure/flow relationships of 2.5, 3.0, 3.5 and 4-mm tracheal tubes were determined at a series of flows from 0.5 to 4 litres/minute. Peak airway and alveolar pressures were then measured at various frequencies and inspiratory:expiratory ratios with the tubes incorporated in a model lung. Large differences between peak airway and alveolar pressures developed when frequency was increased or inspiratory time decreased; the differences were greatest with the smaller tubes. Shortening expiratory time by increasing the frequency or altering the inspiratory:expiratory ratio resulted in increased end-expiratory pressure because of incomplete emptying of the lung.

### Key words

Ventilation; mechanical.

Airway; resistance.

Equipment; tubes, tracheal.

Anaesthesia; paediatric, neonate.

There are many reports of pulmonary barotrauma in neonates ventilated with high peak airway pressures.<sup>1–3</sup> However, the site of the airleak is believed to be at the junction of the small airways and alveoli and so it is likely that the incidence is related more closely to alveolar than to airway pressure.<sup>4</sup>

Airway pressure may be sensed within the ventilator or at the patient Y-piece during mechanical ventilation. The peak pressure at the ventilator end of the inspiratory tube will be higher than that at the Y-piece because of the inspiratory tube flow resistance. The peak pressure recorded at the ventilator end of the expiratory tube will, however, tend to reflect that at the Y-piece because of the absence of flow down the expiratory tube during inspiration. However, the difference in pressure between the two ends of the tracheal tube is much greater than the pressure drop along the ventilator tubing and may result in significant differences between peak airway and alveolar pressures. Furthermore, if the expiratory time is inadequate, a positive end-expiratory pressure may develop in the alveoli and so may modify the relationship between maximum, minimum and mean airway and alveolar pressures. These relationships have been studied by theoretical analysis and by experiments on a model lung, using frequencies between 10 and 200 breaths/minute.

### Methods

#### Theoretical calculations

The tidal volume ( $V_T$ ) necessary to maintain normocapnia in a 3-kg neonate, at frequencies ( $f$ ) of 10–200 breaths per minute (bpm) were first calculated assuming an alveolar ventilation rate ( $\dot{V}_A$ ) of 460 ml/minute<sup>5</sup> and a deadspace of 6 ml. The peak airway pressures required to generate these tidal volumes were then calculated. The pressure component ( $P_c$ ) from elastic recoil was calculated from the formula:—

$$P_c = \frac{V_T}{C}$$

where  $C$  is the total thoracic compliance. The pressure component due to airways resistance ( $P_R$ ) was then calculated from the formula:—

$$P_R = R \times \dot{V}_I$$

where  $R$  is the airway resistance and  $\dot{V}_I$  is the inspiratory flow. Constant inspiratory flow was assumed and the flow calculated by dividing the tidal volume by the inspiratory time, assuming an inspiratory:expiratory (I:E) ratio of 1:1. Peak airway pressures were calculated for combinations of lung compliance of 5 and 1 ml/cmH<sub>2</sub>O and resistances of 50 and 200 cmH<sub>2</sub>O/litres/second at frequencies of 10 to 200 breaths/minute.

T.R. Tipping, MBBCh, FFARCSI, Senior Registrar, M.K. Sykes, MA, MB, BChir, FFARCS, FFARACS (Hon), FFA (SA) (Hon), Nuffield Professor of Anaesthetics, Nuffield Department of Anaesthetics, Radcliffe Infirmary, Oxford OX2 6HE, UK.

Accepted 30 October 1990.

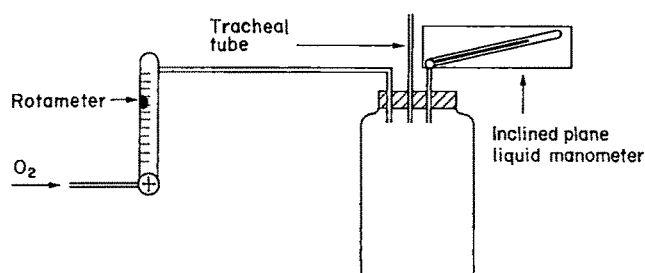


Fig. 1. Apparatus used to measure tracheal tube resistance.

#### Measurement of tracheal tube resistance

The resistances of the neonatal tracheal tubes used in the study were measured using the apparatus shown in Fig. 1. The Rotameter was first calibrated for oxygen flow of 500–4000 ml/minute by passing the gas through a previously calibrated dry gas meter for known periods of time. The Rotameter was then used to deliver various flows of oxygen into a glass bottle with three openings. One was connected to the Rotameter, the second to an inclined plane liquid manometer and the third to 10-cm long Portex tracheal tubes of 2.5, 3.0, 3.5 and 4.0 mm internal diameters. The pressure drop down each tube was measured at flows from 500 to 4250 ml/minute.

#### Model lung experiments

Airway and alveolar pressures were measured with a ventilator model lung system using the four sizes of tracheal tube and tidal volumes and frequencies used in the original calculations. Two model lungs were constructed with compliances of 5 and 1 ml/cmH<sub>2</sub>O (Fig. 2). The lung consisted of a glass bottle filled with copper gauze to ensure an isothermal compression. The ventilator was driven by a high pressure oxygen source (400 kPa) and produced a square wave inspiratory flow pattern with a variable inspiratory:expiratory time ratio, using a T-piece system with an electronically-controlled solenoid expiratory valve. The inspiratory flow and I:E ratios were recorded with a Fleisch 00 pneumotachograph transducer situated between the ventilator Y-piece and the tracheal tube connector. The pneumotachograph was calibrated using oxygen against flows measured by the gas meter. Airway pressure was measured from a right-angled connector situated in the tubing just proximal to the tracheal tube, and 'alveolar pressure' from the inside of the model lung. The gas flow and pressures were recorded on a Lectromed heated stylus recorder.

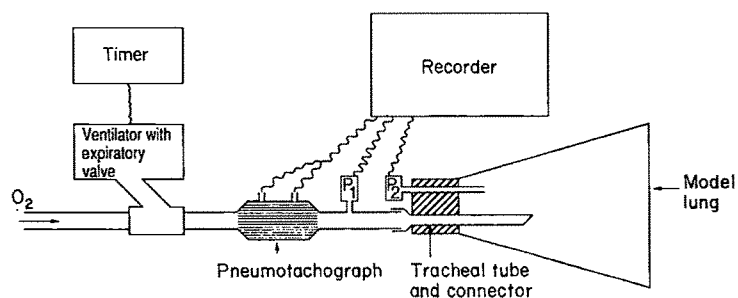


Fig. 2. Ventilator model lung system used to measure flows and pressures.

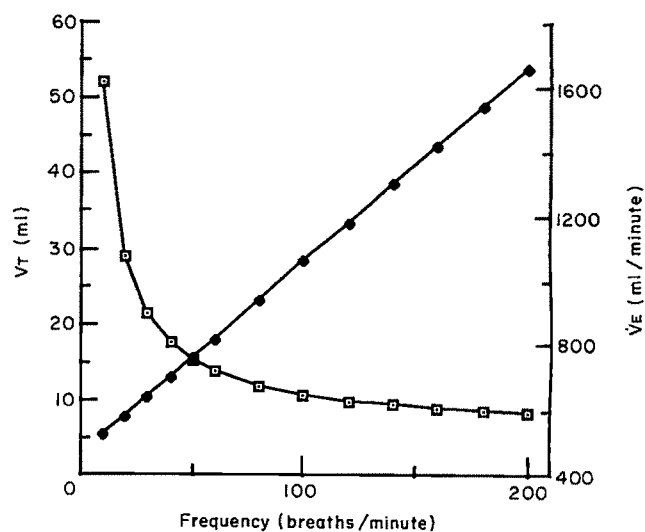


Fig. 3. Calculated tidal volumes ( $V_T$ ) and minute volume ( $\dot{V}_E$ ) required to maintain normocapnia at different frequencies of ventilation. —□—,  $V_T$ ; —◆—,  $\dot{V}_E$ .

The inspiratory flow was adjusted to produce the tidal volume required to maintain an alveolar ventilation of 460 ml/minute at each frequency and I:E ratio. Tidal volume was adjusted by calculating the difference between end-expiratory and end-inspiratory alveolar pressures needed to produce the required tidal volume with each compliance and then adjusting the gas flow to produce this pressure difference within the model lung.

Recordings were made with 2.5, 3.0, 3.5 and 4.0 mm internal diameter tracheal tubes, and model lung compliances of 5 and 1 ml cmH<sub>2</sub>O at frequencies of 10, 20, 40, 80 and 160 breaths per minute and I:E ratios of 1:2, 1:1 and 2:1.

## Results

#### Theoretical calculations

Figure 3 shows the tidal volumes which would be required to maintain an alveolar ventilation of 460 ml/minute at respiratory frequencies of 10 to 200 breaths/minute, assuming a deadspace of 6 ml. Tidal volume decreases markedly as frequency is increased from 10 to 60 breaths/minute, but at higher frequencies (when tidal volume approaches the deadspace) there is little change. Minute volume increases linearly with frequency.

Figures 4 and 5 show the calculated peak airway pressures which would be generated at each frequency with

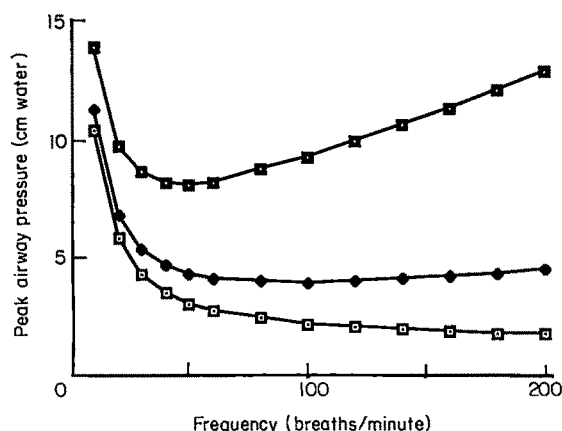


Fig. 4. Calculated peak airway pressures resulting from the delivery of tidal volumes shown in Figure 3 into model lungs with a compliance of 5 ml/cmH<sub>2</sub>O (C5) and resistances of zero (R0) □, 50 (R50) ◆ and 200 (R200) ■ cmH<sub>2</sub>O/litres/second.

compliances of 5 and 1 ml/cmH<sub>2</sub>O, and linear airway resistances of 0, 50 and 200 cmH<sub>2</sub>O/litres/second. The curves for R = 0 show the peak alveolar pressure generated at each frequency by the compliance component alone. This decreases rapidly with increasing frequency at the lower frequencies but changes little at the higher frequencies when tidal volume approaches the volume of the dead-space. The pressure drop from airway resistance increases with frequency as minute volume and inspiratory flow increase, so that peak airway pressure is at a minimum at approximately 50 breaths/minute with the 5 ml/cmH<sub>2</sub>O compliance, and approximately 120 breaths/minute with the 1 ml/cmH<sub>2</sub>O compliance.

#### Measurement of tracheal tube resistance

The pressure-flow plots are nonlinear and are shown in Figure 6.

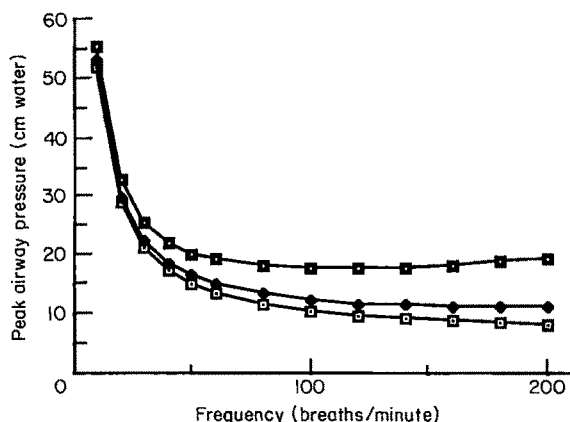


Fig. 5. Calculated peak airway pressures resulting from the delivery of the tidal volumes shown in Figure 3 into model lungs with a compliance of 1 ml/cmH<sub>2</sub>O (C1) and resistances of zero (R0) □, 50 (R50) ◆ and 200 (R200) ■ cmH<sub>2</sub>O/litres/second.

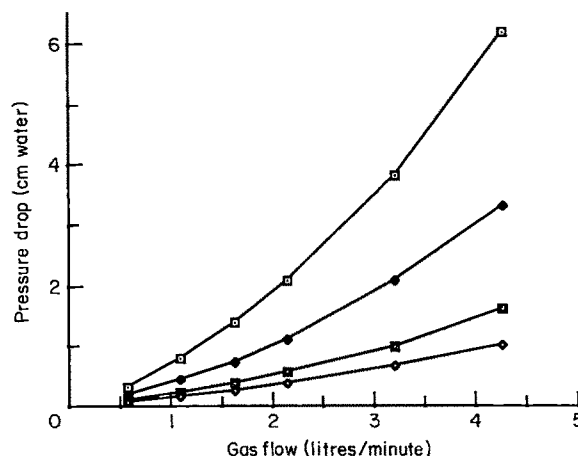


Fig. 6. Measured pressure drop along tracheal tubes with internal diameters of □, 2.5 mm; ◆, 3.0 mm; ■, 3.5 mm; ◇, 4.0 mm at various gas flows.

#### Model lung experiments

The model lung did not incorporate a resistance unit to simulate airway resistance, so all the resistance was from the tracheal tube.

Figure 7 shows the peak airway pressures measured at different frequencies, with the four sizes of tube tested with compliances of 5 and 1 ml/cmH<sub>2</sub>O. Both these sets of measurements were made with an I:E ratio of 1:1 and are compared with the calculated peak alveolar pressures at each frequency. It is apparent that at low frequencies most of the peak airway pressure is the result of the compliance component whilst at higher frequencies the resistance component becomes more important.

Figure 8 shows how the peak airway pressure at each frequency varies with I:E ratio for the 2.5-mm internal diameter tube. As would be expected, the pressure drop along the tube increases as the flow is increased with increasing frequency and with a decrease in the proportion of inspiratory time.

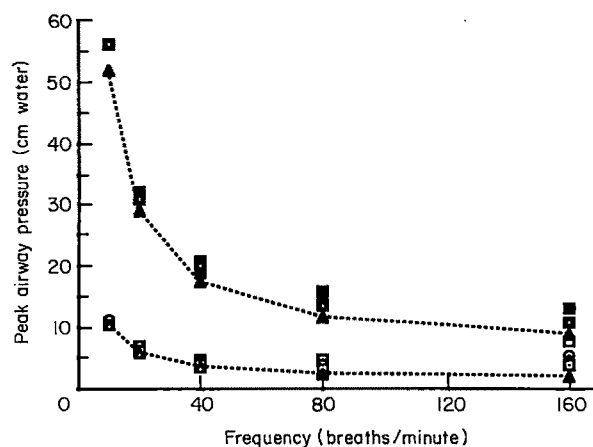


Fig. 7. Peak airway pressures recorded with tracheal tubes of various internal diameters and model lung compliances of 5 and 1 ml/cmH<sub>2</sub>O (C5, C1). The dotted lines show the peak airway pressures which would be generated in the absence of any airway resistance. □, 2.5 mm C5; ○, 3.0 mm C5; △, 3.5 mm C5; ◇, 4.0 mm C5; ■, 2.5 mm C1; ●, 3.0 mm C1; ▲, 3.5 mm C1; ◆, 4.0 mm C1; ....△.... from Fig. 4; ....▲..... from Fig. 5.

**Table 1.** Measured end-expiratory alveolar pressure (cmH<sub>2</sub>O) related to tube size (mm), breathing frequency (breaths/minute) and inspiratory:expiratory time ratio (I:E) with compliance of 5 ml/cmH<sub>2</sub>O.

Frequency	I:E	Tube diameter (mm)			
		2.5	3.0	3.5	4.0
40	1:2	0	0	0	0
	1:1	0	0	0	0
	2:1	0.5	0.5	0	0
80	1:2	0.8	0.4	0	0
	1:1	1.0	0.8	0	0.2
	2:1	2.4	1.8	1.2	0.8
160	1:2	2.0	1.6	1.0	0.6
	1:1	3.0	2.0	1.4	1.0
	2:1	13.0	5.2	2.8	2.4

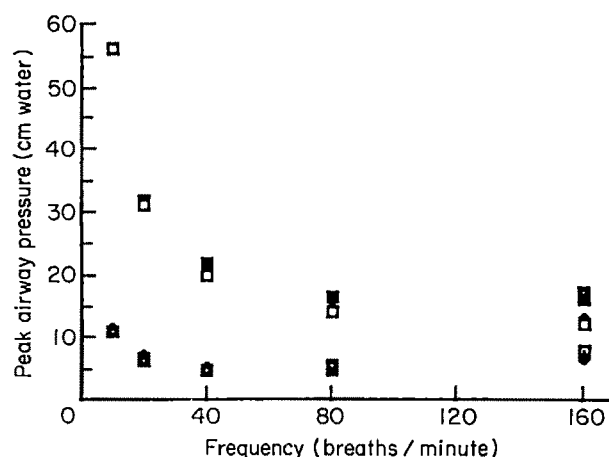
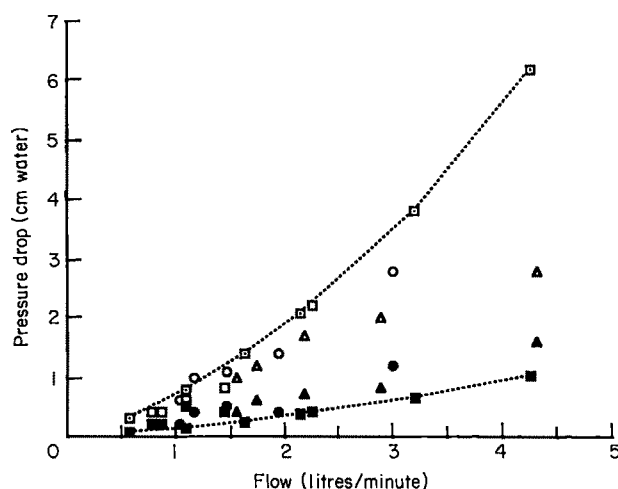
At low frequencies, when the inspiratory flow is low, there is little difference in the peak airway pressure with different I:E ratios. However, at 160 breaths/minute, when flow is increased there is a large difference between the 1:2 and 2:1 ratios.

Figure 9 shows the pressure drop down the tube measured in the model lung plotted against various flows generated by changing I:E ratios and frequency. The values for the 2.5- and 4-mm internal diameter tubes are compared with the pressure drop down that tube measured with continuous flow, as shown in Figure 6. There was a reasonable agreement between the values for pressure drop down the tube when measured by the two methods.

### Discussion

As shown in Figure 3, increasing the respiratory rate enables a constant alveolar ventilation to be maintained with a decreasing tidal volume, thereby reducing the peak airway and alveolar pressures. However, as the tidal volume decreases towards the deadspace volume the proportion of deadspace ventilation increases so that minute volume must be increased linearly to maintain a constant alveolar ventilation and arterial  $P_{CO_2}$ . If the I:E ratio is fixed it is necessary to generate higher inspiratory flows in order to transfer the increased minute volume to the alveoli at the higher frequencies. This must result in a greater pressure decrease along the tracheal tube at higher frequencies (Figs 4 and 5). Thus, as frequency is increased the component of airway pressure due to compliance decreases whilst that due to tube (and airway) resistance increases. This results in a U-shaped curve; the minimum peak airway pressure occurs at approximately 50 breaths/minute with the 5 ml/cmH<sub>2</sub>O compliance and 50 cmH<sub>2</sub>O/litres/second resistance combination, and at around 120 breaths/minute with the same tube resistance but 1 ml/cmH<sub>2</sub>O compliance. Similar findings have been documented in adults.<sup>6,7</sup>

If the proportion of inspiratory time ( $T_i$ %) is reduced, flow must be further increased, so increasing the pressure drop along the tube (Fig. 8). Obviously, the pressure drop will be greater with a narrow tube than with a wide one (Fig. 7) and the nonlinearity of the tube resistance will accentuate the pressure drop at high flows (Figs 6 and 9). It is thus apparent that whilst peak airway pressure will provide a reasonable estimate of peak alveolar pressure at low frequencies, there will be an increasing discrepancy between peak airway pressure and peak alveolar pressure

**Fig. 8.** Peak airway pressures recorded with compliances of 5 and 1 ml/cmH<sub>2</sub>O (C5, C1) and three inspiratory:expiratory time ratios (1:1, 1:2 and 2:1). □, C5 1:1; ◆, C5 1:2; ■, C5 2:1; ◇, C1 1:1; ■, C1 1:2; □, C1 2:1.**Fig. 9.** Comparison of pressure drop along 2.5 and 4 mm tracheal tubes measured during steady flow (dotted lines, data from Fig. 6) and pressure drops measured in model lung with various I:E ratios (1:1, 1:2 and 2:1). ○, 2.5 mm 1:1; △, 2.5 mm 1:2; □, 2.5 mm 2:1; ●, 4.0 mm 1:1; ▲, 4.0 mm 1:2; ■, 4.0 mm; ....□....., 2.5 mm from Fig. 6; ....■....., 4.0 mm from Fig. 6.

as frequency is increased.<sup>7</sup> The difference between peak airway and alveolar pressure will be increased further if the tube is narrow or the proportion of inspiratory time is decreased.

An additional factor which causes peak airway and alveolar pressures to increase with increasing frequency is the development of an end-expiratory pressure, as a result of incomplete emptying of the lung. The pattern of emptying is approximately exponential and determined by the time constant ( $T$ ) which in turn depends on the compliance ( $C$ ) and the resistance ( $R$ ):—

$$T = C \times R$$

For a lung where  $C = 5$  ml/cmH<sub>2</sub>O and  $R = 50$  cmH<sub>2</sub>O/litres/second.  $T = 0.25$  second, so 95% of the tidal volume should have been expired in 0.75 second (three time constants). However, with a similar compliance and a resistance of 200 cmH<sub>2</sub>O litres/second this time would have been increased to 3 seconds. The inspiratory airflows used in the present experiments varied from 0.78 to 4.3 litres/minute during inspiration (0.013–0.712 litres/second) but

because of the exponential pattern of expiration, peak expiratory flows were somewhat higher. However, at 4 litres/minute flow, the pressure drop along the 2.5 mm tube was 5.5 cmH<sub>2</sub>O. The resistance at this flow is therefore 82.5 cmH<sub>2</sub>O/litres/second and the time constant with the 5 ml/cmH<sub>2</sub>O compliance is 0.41 second. Thus, the lung would not be expected to empty during the expiratory time available at the higher frequencies.

In the present experiments there was no increase in end-expiratory alveolar pressure with the 1 ml/cmH<sub>2</sub>O compliance with any of the tubes and I:E ratios tested. However, with the 5-ml/cmH<sub>2</sub>O compliance, a positive end-expiratory alveolar pressure was seen at 40 breaths/minute with the 2.5- and 3.0-mm tubes and at 80 breaths/minute with the two larger tubes. The increase was greatest with the 2:1 ratio and frequency of 160 breaths/minute where the end-expiratory pressure was 13 cmH<sub>2</sub>O with the 2.5-mm tube. This resulted in a peak alveolar pressure of 14.8 cmH<sub>2</sub>O and a peak airway pressure of 17.0 cmH<sub>2</sub>O. It is possible that *in vivo* a difference between inspiratory and expiratory impedances may accentuate this effect.<sup>8</sup>

These experiments show that the increasing inspiratory and expiratory flows associated with the increased proportion of deadspace ventilation at higher frequencies of ventilation cause large discrepancies between airway and alveolar pressures. A constant deadspace has been assumed and if there is a reduction in anatomical and physiological deadspace with tidal volume, as has been described in dogs<sup>9</sup> and adults,<sup>10</sup> the magnitude of the effects would be reduced. However, the airway resistance has also been ignored and if this is added to the tube resistance the effect would be accentuated.

Studies of the pressure-flow relationships of tracheal tubes during high frequency ventilation have shown that the pressure drop measured when the tubes were inserted into the trachea of anaesthetised paralysed dogs was 30–50% higher than when measured *in vitro*. The pressure drops down the tracheal tubes were also shown to be nonlinearly related to peak flows and were dependent on tidal volume.<sup>11</sup> It is thus possible that the pressure drops down the tracheal tubes measured in our experiments may underestimate the differences observed in the patients.

Bench tests by Synnott *et al.*<sup>7</sup> showed that although there

were large increases in the pressure displayed on the ventilator pressure gauge when respiratory frequency was increased from 20 to 120 breaths/minute, the increase in peak intratracheal pressure was much less. He also found that increasing inspiratory time from 20 to 80% of the respiratory period caused the ventilator to display a decrease in peak pressure even though distal airway pressure increased. Our findings thus confirm his observations.

## References

1. GREENOUGH A, DIXON AK, ROBERTSON NRC. Pulmonary interstitial emphysema. *Archives of Disease in Childhood* 1984; **59**: 1046–51.
2. OGATA ES, GREGORY GA, KITTERMAN JA, PHIBBS RH, TOOLEY WH. Pneumothorax in the respiratory distress syndrome: incidence and effect on vital signs, blood gases, and pH. *Pediatrics* 1976; **58**: 177–83.
3. BERG TJ, PAGTAKHAN RD, REED MH, LANGSTON C, CHERNICK V. Bronchopulmonary dysplasia and lung rupture in hyaline membrane disease: influence of continuous distending pressure. *Pediatrics* 1975; **55**: 51–4.
4. MACKLIN CA. Transport of air along sheaths of pulmonic blood vessels from alveoli to mediastinum. *Archives of Internal Medicine* 1939; **64**: 913–26.
5. GOLDSMITH JP, KAROTKIN EH. *Assisted ventilation of the neonate*, 2nd edn. Philadelphia: W.B. Saunders, 1988: 436.
6. CHAKRABARTI MK, GROUNDS RM, SWENZEN GO, WHITWAM JG. Relationship between frequency of ventilation, airways and pulmonary artery pressures, cardiac output and tracheal tube deadspace. *Acta Anaesthesiologica Scandinavica* 1986; **30**: 678–84.
7. SYNNOTT A, WREN WS, DAVENPORT J. Peak intratracheal pressure during controlled ventilation in infants and children. *Anaesthesia* 1987; **42**: 719–26.
8. SIMON BA, WEINMANN GG, MITZNER W. Mean airway pressure and alveolar pressure during high-frequency ventilation. *Journal of Applied Physiology* 1984; **57**: 1069–78.
9. WILLIAMS MH, RAYFORD CM. Effect of variation of tidal volume on size of physiological dead space in dogs. *Journal of Applied Physiology* 1956; **9**: 30–32.
10. CHAKRABARTI MK, GORDON G, WHITWAM JG. Relationship between tidal volume and deadspace during high frequency ventilation. *British Journal of Anaesthesia* 1986; **58**: 11–17.
11. GAVRIELY N, SOLWAY J, LORING SH, BUTLER JP, SLUTSKY AS, DRAZEN JM. Pressure-flow relationships of endotracheal tubes during high-frequency ventilation. *Journal of Applied Physiology* 1985; **59**: 3–11.



## Percutaneous dilational tracheostomy

### A bedside procedure on the intensive care unit

A. BODENHAM, R. DIAMENT, A. COHEN AND N. WEBSTER

#### Summary

*Tracheostomy is performed commonly to aid weaning from assisted ventilation in patients who require intensive care. The procedure carries a significant mortality and morbidity, in part due to problems in moving unstable patients. We report the use of a bedside percutaneous tracheostomy technique for use in adult patients who require intensive care. The procedure was successful in 19 of 20 cases. The one failure, in the first case, resulted from transection of a superficial midline vein. The lack of significant haemorrhage was notable in all other cases. The procedure has proved to be efficient and cost-effective in this unit and has now replaced conventional surgical tracheostomy in this group of patients.*

#### Key words

*Surgery; tracheostomy.*

*Equipment; percutaneous tracheostomy kit.*

Tracheostomy is performed commonly in patients who require intensive care although clinicians vary in their indications and preferred timing of the procedure when it is performed to aid weaning from assisted ventilation.<sup>1</sup> The procedure has significant mortality and morbidity, due in part to problems in moving unstable ventilated patients to and from the operating theatre. Many units perform tracheostomy by conventional surgical techniques on the intensive care unit (ICU).<sup>2</sup> We have explored the use of the 'Ciaglia' percutaneous dilational tracheostomy kit<sup>3</sup> for the formation of adult tracheostomies in the ICU. The technique originates from the USA and has not been reported previously in the UK.

#### Method

Patients who required tracheostomy gave consent in the usual fashion. The first four procedures were carried out in the operating theatre; thereafter, procedures were performed in the ICU. Patients were selected initially on the basis of apparently normal neck anatomy, normal stature, no bleeding tendency and an easily palpable cricoid cartilage and upper trachea. Surgeons and all conventional equipment were present at each of the first eight procedures. No eligible adult patient was considered unsuitable for the technique in the 8-month study period.

An anaesthetist other than the operator was responsible for airway care and anaesthesia, which comprised infusions of alfentanil and propofol. Muscle relaxation was provided

by atracurium, as required. The tracheal tube was withdrawn under direct laryngoscopy until the cuff lay within the larynx and the lungs were ventilated with 100% oxygen.

The head and neck were extended as for conventional tracheostomy. A 1.5–2 cm transverse incision was made midway between the cricoid cartilage and sternal notch. A 14 g cannula was inserted into the trachea between the first and second tracheal rings after blunt dissection of the pretracheal fascia. A 'J' guide wire was introduced into the trachea through the cannula and the tracheostomy dilated sequentially to 36 FG (12 mm) using specially designed plastic dilators (Cook Critical Care Ltd, Letchworth, Herts) (Fig. 1). The appropriate tracheostomy tube (8 or 9 mm internal diameter; Portex Ltd, Hythe, Kent) was then inserted over a smaller dilator. Air leaks through the dilation tract during the procedure were controlled by digital pressure.

#### Results

The procedure was successful in 19 of 20 patients whose ages ranged from 23 to 82 years. Patients had received assisted ventilation on the ICU for a range of 4–22 days. The only failure was in the first case, when blunt dissection was inadequate and a midline superficial vein was transected by the needle and guidewire. Bleeding increased with dilation and the procedure was abandoned in favour of conventional surgery. There was minimal bleeding in subsequent cases. Six patients had significant liver dysfunction,

A. Bodenham, FFARCS, Senior Registrar Intensive Care, R. Diament, FRCS, Surgical Registrar, A. Cohen, FFARCS, Consultant Intensive Care, N. Webster, PhD, FFARCS, Consultant Intensive Care, St James's Hospital, Leeds LS9 7TF.

Accepted 9 January 1991.

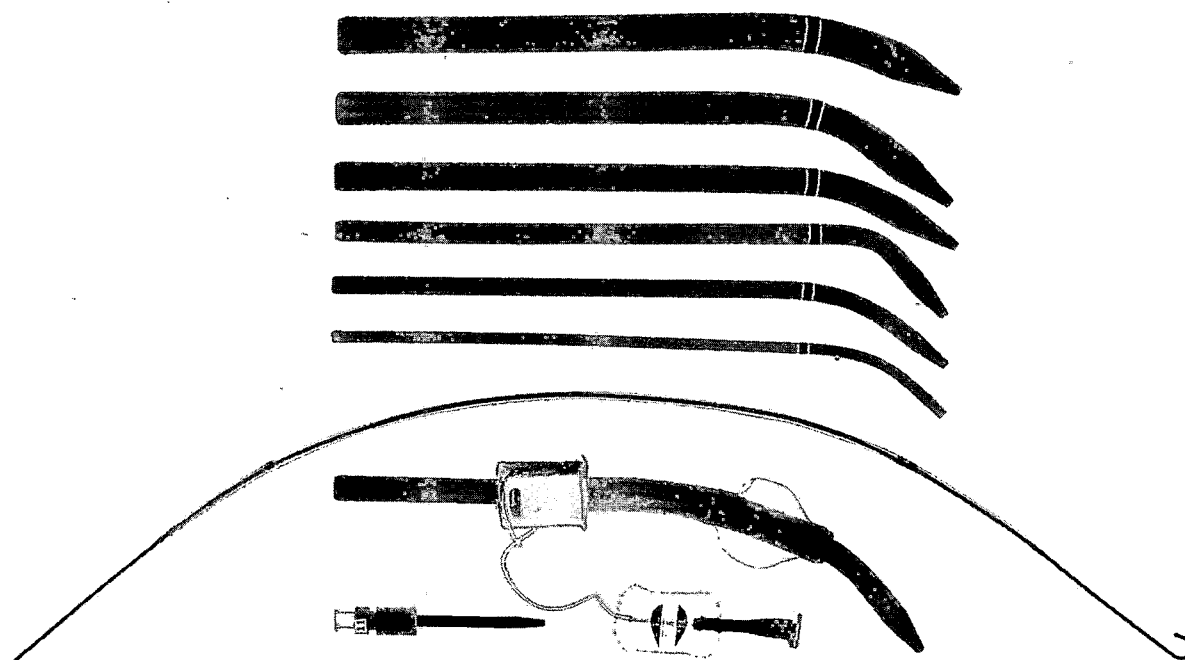


Fig. 1. 'Ciaglia' percutaneous tracheostomy kit with 8 mm Portex tracheostomy tube mounted on 24FG dilator.

with abnormal coagulation studies; fresh frozen plasma was infused during the procedure in these cases. In one patient, who had undergone conventional tracheostomy 6 months previously, the tracheostomy was reformed successfully through the old scar. All tubes were changed routinely at 7 days without difficulty. The earliest tube change was performed 24 hours after insertion, without difficulties, after insertion of a double-lumen bronchial tube was required for thoracic surgery.

Minor difficulties were encountered in one patient when using a Shiley tracheostomy tube, which did not conform to the shape of the dilator; the problem was resolved by using a softer Portex tube. An 8-mm tube could not be inserted in one female patient but a 7.5-mm tube was introduced successfully.

### Discussion

A number of procedures, formerly the exclusive preserve of surgeons, may now be carried out by percutaneous dilatational techniques. We have confirmed that the 'Ciaglia' dilatational tracheostomy technique is safe and effective. Our results demonstrate that it is a suitable technique for intensivists without formal surgical training, although surgical colleagues can provide invaluable assistance when learning the technique and should be aware that the procedure has been adopted. The procedure is best performed with an assistant who helps to stabilise the guidewire while dilators are changed. We estimate that costs are halved in comparison to conventional tracheostomy performed in the operating theatre, despite the relatively high cost of the disposable dilators.

The presence of superficial midline veins provides one potential bleeding site in this procedure. The one failure, when blunt dissection was too superficial, highlighted the presence of such veins, which were identified in a number of

subsequent cases. With our further experience, we would probably now have removed the dilator and achieved haemostasis by pressure or tying off the vein. Once bleeding stopped we would restart the dilatational technique from a site immediately lateral to the bleeding point. Any bleeding is effectively eliminated by compression when the tracheostomy tube is *in situ*. The lack of bleeding after this<sup>2-4</sup> and other dilatational techniques<sup>5</sup> confirms this advantage of the technique, particularly in the presence of a bleeding tendency. The tract fits tightly round a tracheostomy tube until 2-3 days later, making re-insertion potentially difficult. It is unclear whether an early change of tracheostomy tube may be more difficult after dilatational techniques than after conventional tracheostomy. Our limited experience with early tube changes suggests that this is not the case. However, the technique may be inappropriate if conventional oral/nasotracheal intubation cannot be performed reliably. We recommend that the use of this technique should be restricted to areas in which medical staff skilled in airway care are present.

There was no mortality associated with the procedure. The incidence of longer term problems such as tracheal stenosis after dilatational techniques is unknown. There is a low frequency of such problems and a high mortality among intensive care patients who require tracheostomy. Consequently, it may be more appropriate to study these complications in a different group of patients.

A number of different guide wire tracheostomy techniques have been reported recently<sup>6,7</sup> but no direct comparison has been made between techniques.

### Acknowledgments

We thank our surgical colleagues in Leeds for their support during the introduction of this technique.

# References

1. MARSH HM, GILLESPIE DJ, BAUMGARTNER AE. Timing of tracheostomy in the critically ill patient. *Chest* 1989; **96**: 190-3.
2. STEVENS DJ, HOWARD DJ. Tracheostomy service for ITU patients. *Annals of the Royal College of Surgeons of England* 1988; **70**: 241-2.
3. CIAGLIA P, FIRSCHING R, SYNIEC C. Elective percutaneous dilatational tracheostomy. A new simple bedside procedure; Preliminary report. *Chest* 1985; **87**: 715-9.
4. COOK PD, CALLANAN VI. Percutaneous dilatational tracheostomy technique and experience. *Anaesthesia and Intensive Care* 1989; **17**: 456-7.
5. HAZARD PB, GARRETT HE, ADAMS JW, ROBBINS ET, AGUILLARD RN. Bedside percutaneous tracheostomy: experience with 55 elective procedures. *Annals of Thoracic Surgery* 1988; **46**: 63-7.
6. TOYE FJ, WEINSTEIN JD. Clinical experience with percutaneous tracheostomy and cricothyroidotomy in 100 patients. *Journal of Trauma* 1986; **26**: 1034-40.
7. SCHACHNER A, OVIL Y, SIDI J, ROGEV M, HEILBRONN Y, LEVY MJ. Percutaneous tracheostomy—A new method. *Critical Care Medicine* 1989; **17**: 1052-6.

## The effect of micropore filters on the aspiration test in epidural analgesia

G. A. CHARLTON AND E. G. LAWES

### Summary

Continuous epidural analgesia may be complicated by intravascular or subarachnoid injection of local analgesic, with disastrous consequences. One of the techniques described to prevent these complications is the aspiration test. It is the current standard of practice to employ a micropore bacterial filter for epidural infusions. We present an *in vitro* study of nine commercially available bacterial filters to determine whether or not the aspiration test could be reliably performed through them. Eight of the nine filters (except the Perifix, Braun) were found to be unreliable if air and liquid were both present in the system (air lock). The role of the aspiration test and the use of micropore filters in epidural analgesia are discussed, as are the implications and possible solutions in practice of this cause of failure of the test.

### Key words

Equipment; bacterial filters.

Anaesthetic techniques, regional; epidural.

Continuous epidural infusions are commonly used for obstetric analgesia, postoperative analgesia and for the control of chronic pain. Hazards of this technique include inadvertent placement or migration of the epidural catheter into the intravascular<sup>1,2</sup> or subarachnoid space,<sup>3,4</sup> with potentially disastrous consequences. The combined incidence of these complications has been reported to be as high as 3–10 per 10 000 epidurals.<sup>5,6</sup> As early as 1933, Dogliotti recommended that an aspiration test be performed and a test dose of local analgesic be given, before injecting drugs through the epidural catheter, so that incorrect catheter position might be excluded.<sup>7</sup>

The use of micropore filters in continuous epidural analgesia was first advocated in 1972.<sup>8</sup> Since then they have been shown to be beneficial in excluding both particulate matter<sup>9,10</sup> and bacterial contamination.<sup>11</sup>

This study aimed to compare, *in vitro*, the effect of various commercially available bacterial filters on the performance of the aspiration test.

### Methods

We studied nine different bacterial filters (Table 1). Although the Posidyne and Ultipor intravenous filter/air eliminators are not designed to filter epidural infusions, we have heard of them being used for this purpose and there-

Table 1. Micropore filters tested.

Filter	Pore size (µm)	Manufacturer
1. Everett E500 filter	0.22	Avon Medicals
2. Encapsulon filter	0.2	Warne/Franklin
3. Millex-GS filter	0.22	Millipore
4. Pall set saver	0.2	Pall
5. Perifix filter	0.2	Braun
6. Portex flat filter	0.2	Portex
7. Sterifix filter	0.2	Braun
8. Posidyne intravenous filter/air eliminator	0.2	Pall
9. Ultipor intravenous filter/air eliminator	0.2	Pall

fore decided to study them. Each filter was tested with the various combinations of air and bupivacaine as listed in Table 2. We believe that this encompassed all possible clinical situations. Attempts were made to aspirate either heparinised blood or 'cerebrospinal fluid' (normal saline stained with blue ink, so that passage of the saline in the catheter could be identified) from a beaker. Both slow, low-suction pressure and fast, high-suction pressure aspiration attempts were made on each filter. Two-ml, 10-ml and 20-ml syringes were used with each study. Attempts were also made to inject with each of the combinations.

G.A. Charlton, BSc, FFARCS, Locum Senior Registrar, E.G. Lawes FFARCS, Consultant, Shackleton Department of Anaesthetics, Southampton General Hospital, Tremona Road, Southampton SO9 4XY.

Accepted 18 October 1990.

Table 2. Test sequences for each filter.

	Catheter contents	Filter contents	Syringe contents	Successful test	
				Aspirate	Inject
A.	Air	Air	Air	9/9	9/9
B.	Air	Air	Bupivacaine	9/9	9/9
C.	Bupivacaine	Bupivacaine	Air	9/9	1/9*
D.	Bupivacaine	Bupivacaine	Bupivacaine	9/9	9/9
E.	Air	Bupivacaine	Air	1/9*	1/9*
F.	Air	Bupivacaine	Bupivacaine	1/9*	9/9
G.	Bupivacaine	Air	Air	9/9	9/9
H.	Bupivacaine	Air	Bupivacaine	9/9	9/9

\*Exceptions in all cases were the Perifix filter (Braun).

All suction attempts were performed by hand. A successful test was deemed to be one in which the test fluid was easily aspirated from a glass beaker a distance of 20 cm into the distal end of a standard 18-gauge epidural catheter.

### Results

There was no difference between blood or coloured saline with respect to the ease with which each could be aspirated into the epidural catheter. Similarly, there were no differences in the way fluid was aspirated when different sized syringes and different aspirating forces were applied. We were unable to disrupt the integrity of any of the filters during the performance of the test sequences. The results obtained with each of the tests are summarised in Table 2. All aspirations and injections were possible with all makes of filter except when the filter was wet and an attempt was made to pass air through it. In this situation an air lock developed and we were unable to aspirate or inject through the filter. The only filter unaffected by the air lock was the Perifix Filter (Braun).

### Discussion

Injection of a large volume of local analgesic solution into either the subarachnoid or intravascular space can have lethal consequences, particularly in the parturient patient. Prince and McGregor<sup>12</sup> have restated that the aspiration test is vital to exclude catheter misplacement. Numerous tests, utilising glucose test strips,<sup>13,14</sup> urine test strips<sup>15</sup> and thiopentone,<sup>16</sup> have been described to assist with the accurate identification of aspirated cerebrospinal fluid (CSF). As little as 0.05 ml of aspirate is needed to confirm the presence of glucose on the glucose test strip.<sup>17</sup> False-positive results have been obtained with both blood<sup>13</sup> and saline<sup>18</sup> and this has led Prince and McGregor<sup>12</sup> to state that CSF aspiration can only be diagnosed with certainty if more fluid is aspirated than is injected. Blood, by virtue of its colour, is more easily identified. Our study has shown that blood and crystalloid solution can be aspirated with equal ease, no matter what syringe size or aspirating pressure is used.

Controversy surrounds the use of micropore bacterial filters in epidural analgesia. Filters have been recommended to exclude contamination of the epidural space by particulate matter,<sup>8,10</sup> including glass fragments from ampoules.<sup>9,19</sup> In an uncontrolled study, James *et al.*<sup>11</sup> showed that, in the presence of a bacterial filter, all catheter

tips were sterile, whilst 5/101 syringes were contaminated by bacteria. Several authors recommend their use to avoid the complication of epidural abscess formation.<sup>20,10</sup> Abouleish and Amortegui<sup>21</sup> state, however, that micropore filters are not necessary for epidural analgesia. They have shown that a filter needle used for aspirating a drug from the glass ampoule was an 'adequate, cheaper, and less cumbersome means of protecting the epidural space'.<sup>22</sup> The fact that epidural abscesses have occurred despite the utilisation of micropore filters,<sup>23,24</sup> led them to perform a double-blind, controlled study on the benefit of filters in obstetric epidural analgesia.<sup>25</sup> They concluded that, providing sound sterile techniques are applied, a micropore filter is not required for healthy obstetric patients. It is our opinion that, until further evidence to the contrary is provided, filters should continue to be used for both obstetric and longer-term epidural infusions.

If we accept that filters should be used and that the aspiration test is essential to exclude catheter misplacement, how then should the aspiration test be performed? Our study has shown that all commonly used bacterial filters, with the exception of the Perifix (Braun), will not allow fluid to be aspirated if there is an air-fluid interface in the system. Thus, the benefit of performing the aspiration test will be negated and a possible false-negative result may be obtained. One obvious solution is to ensure that only Perifix (Braun) filters are utilised. A more practical solution would be to ensure that no airlock is present before performing the aspiration test. If, before catheter placement, the patency of the filter and catheter are tested with air, rather than with saline/local analgesic, the situation in which air can enter the wet catheter or filter during insertion and create an airlock would be avoided. This would ensure the accuracy of the initial aspiration test. For repeated top-up doses, however, this would not be possible. Air can enter the system during syringe disconnection and reconnection. In this situation it has been suggested that 1.5 ml of saline be injected through the filter/catheter system before performing the test.<sup>12</sup> This will eliminate any airlock as well as clear any blockage in the catheter, which may also cause a false-negative test result. If filters were eliminated from the system, either entirely or only for the performance of the aspiration test, the problem of air-locking would be excluded. We believe that, because of the risks (although small) of neurological or infective injury, discontinuation of the use of micropore filters is ill-advised.

Although performance of the aspiration test and administration of a test dose will identify the majority of incorrect catheter positions, no test is infallible. The injection of fractionated doses of local analgesic should, therefore, remain the standard of practice.

### Acknowledgments

We thank the individual companies for supplying the filters used for testing.

### References

1. RAVINDRAN R, ALBRECHT W, MCKAY M. Apparent intravascular migration of epidural catheter. *Anesthesia and Analgesia* 1979; **58**: 252-3.
2. ZEBROWSKI ME, GUTSCHE BB. More on intravascular migration of an epidural catheter. *Anesthesia and Analgesia* 1979; **58**: 531.

3. KALAS DB, HEHRE FW. Continuous lumbar peridural anesthesia in obstetrics VIII: further observations on inadvertent lumbar puncture. *Anesthesia and Analgesia* 1972; **51**: 192-5.
4. ROBSON JA, BRODSKY JB. Latent dural puncture after lumbar epidural block. *Anesthesia and Analgesia* 1977; **56**: 725-6.
5. DAWKINS CJM. An analysis of the complications of extradural and caudal block. *Anaesthesia* 1969; **24**: 554-63.
6. CRAWFORD JS. Some maternal complications of epidural analgesia for labour. *Anaesthesia* 1985; **40**: 1219-25.
7. DOGLIOTTI AM. Segmental peridural anesthesia. *American Journal of Surgery* 1933; **20**: 107-18.
8. DESMOND J. The use of micropore filters in continuous epidural anaesthesia. *Canadian Anaesthetists' Society Journal* 1972; **19**: 97-100.
9. TURCO S, DAVIS NM. Glass particles in intravenous injections. *New England Journal of Medicine* 1972; **287**: 1204-5.
10. CRAWFORD JS, WILLIAMS ME, VEALES S. Particulate matter in the extradural space. *British Journal of Anaesthesia* 1975; **47**: 807.
11. JAMES FM, GEORGE RH, NAIEM H, WHITE GJ. Bacteriologic aspects of epidural analgesia. *Anesthesia and Analgesia* 1976; **55**: 187-90.
12. PRINCE G, MCGREGOR D. Obstetric epidural test doses—a reappraisal. *Anaesthesia* 1986; **41**: 1240-50.
13. BERRY A. Test for spinal fluid. *Anaesthesia* 1958; **13**: 100-1.
14. ACKERMAN WE, JUNEJA MM, KACZOROWSKI DM. Identification of cerebrospinal fluid. *Anaesthesia* 1988; **43**: 903.
15. REISNER LS. Epidural test solution or spinal fluid? *Anesthesiology* 1976; **44**: 451.
16. CATTERBERG J. Local anesthetic vs. spinal fluid. *Anesthesiology Review* 1989; **16**: 48-51.
17. JUNEJA MM, ACKERMAN WE. Identification of epidural catheter migration by routine testing of the catheter aspirate. *Anesthesiology Review* 1989; **16**: 48-51.
18. SADDLER JM. Falsely positive reaction to glucose test strips. *Anaesthesia* 1985; **40**: 601.
19. FURGANG FA. Glass particles in ampules. *Anesthesiology* 1974; **41**: 525.
20. NORTH JB, BROPHY BP. Epidural abscess: a hazard of spinal epidural anaesthesia. *Australia and New Zealand Journal of Surgery* 1979; **49**: 484-5.
21. ABOULEISH E, AMORTEGUI AJ. Millipore filters are not necessary for epidural block. *Anesthesiology* 1981; **55**: 604.
22. ABOULEISH E, ORIG T, AMORTEGUI AJ. Bacteriologic comparison between epidural and caudal techniques. *Anesthesiology* 1980; **53**: 511-4.
23. CRAWFORD JS. Pathology in the extradural space. *British Journal of Anaesthesia* 1975; **47**: 412-5.
24. SAADY A. Epidural abscess complicating thoracic epidural analgesia. *Anesthesiology* 1976; **44**: 244-6.
25. ABOULEISH E, AMORTEGUI AJ, TAYLOR FH. Are bacterial filters needed in continuous epidural analgesia for obstetrics? *Anesthesiology* 1977; **46**: 351-4.

## Forum

### Clinical evaluation of a spinal catheter technique in femoro-popliteal graft surgery

L. M. M. Morrison, BSc, FFARCSI, Research Fellow, J. H. McClure, BSc, FCAnaes, Consultant, J. A. W. Wildsmith, MD, FCAnaes, Consultant/Senior Lecturer, Department of Anaesthetics, Royal Infirmary of Edinburgh, Lauriston Place, Edinburgh EH3 9YW.

#### Summary

*The use of a 24-gauge catheter for continuous spinal anaesthesia was evaluated in 20 patients undergoing femoro-popliteal graft surgery for occlusive disease. The catheter was inserted through either a Tuohy or Quincke-tip needle and isobaric amethocaine used for the initial injection. In five patients identification of the subarachnoid space was not straightforward, but clear difficulty with catheter insertion was encountered in only one. The range of blocks seen was wider than expected, but it was adequate for surgery 15 minutes after injection in 16 of the 20 patients. In another three the injection of a small dose of hyperbaric bupivacaine produced the necessary extension of block. Two patients (10%) required a general anaesthetic, one because of grossly inadequate spread of solution, the other because the catheter kinked and prevented injection of a second dose after the start of surgery.*

#### Key words

*Anaesthetic technique, regional; spinal.  
Equipment; catheters, intrathecal.*

Recent developments in plastic technology have made available fine bore catheters that have reawakened interest in continuous spinal anaesthesia. In theory this should allow titration of drug to provide precise control of the extent and duration of block with much smaller doses of local anaesthetic drug than in extradural blocks and without significant risk of postlumbar puncture headache. The technique would seem to hold particular attraction in high risk patients undergoing relatively prolonged surgery to the lower limbs, such as arterial reconstruction. This report describes the evaluation of a 24-gauge catheter for spinal anaesthesia in patients undergoing femoro-popliteal bypass surgery.

#### Method

After ethics committee approval was obtained 20 ASA 2 patients (mean age 69 years, range 55–78) who were to undergo femoro-popliteal bypass surgery for occlusive peripheral vascular disease were recruited into the study. Premedication was with either morphine 5 mg and atropine 0.6 mg intramuscularly or temazepam 10–20 mg orally. After venous access was established, the lumbar region was prepared aseptically with the patient in the right lateral position. Lumbar puncture was performed usually at the third lumbar interspace, but the second interspace was preferred in one patient. A 20-gauge Tuohy needle was used in 11 patients and a Quincke point needle was used in the remaining nine. The needle was inserted with the bevel facing laterally and it was turned cephalad for insertion of the catheter when the subarachnoid space was entered. On obtaining free flow of cerebrospinal fluid (CSF) from the needle, a 24-gauge spinal catheter (Preferred Medical Products, Canada) with a stylet was threaded through the needle. Once the catheter tip reached the needle bevel the

stylet was withdrawn as 3 cm of catheter was introduced simultaneously into the subarachnoid space. CSF was aspirated to verify correct location of the catheter. Ease of identification of the subarachnoid space and of catheter insertion were noted as was the occurrence of any paraesthesia. The patient was then returned to the supine posture and 1.5 ml of 1% isobaric amethocaine (obtained through I.D.I.F. Ltd London) was injected. Difficulty in making the first or subsequent injection of local anaesthetic was also noted. The level of pinpoint analgesia and motor block by the Bromage scale were monitored every 5 minutes for 15 minutes after administration of the first dose to ensure that a block adequate for surgery was obtained. If the block was inadequate further local anaesthetic was administered and its effect reassessed. Subsequent injections of local anaesthetic were given to prolong the block as necessary.

All patients received 1–2 mg of intravenous midazolam at intervals to ensure sleep during the procedure and oxygen was administered by facemask. Routine monitoring of electrocardiogram, blood pressure and oxygen saturation was employed. Hypotension was treated with 3 mg increments of intravenous ephedrine.

The catheter was left *in situ* overnight as is our usual practice in vascular patients and removed the following morning by the anaesthetist. Any difficulty with removal was noted and each patient was visited daily for at least 3 days. They were specifically questioned about the presence of any headache and its nature, backache, paraesthesia or any other sequelae related to the anaesthetic.

#### Results

Difficulty in identification of the subarachnoid space was encountered in two of the 11 patients using the Tuohy needle and three of the nine with the Quincke needle.

Accepted 30 October 1990.



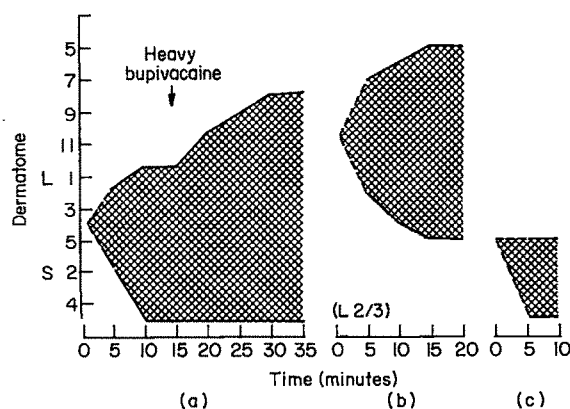


Fig. 1. Dermatome spread of intrathecal amethocaine via a 24-gauge catheter.

Catheter insertion was difficult in only one patient; the Tuohy needle was inserted with ease in that case. A mild transient paraesthesia was noted during catheter insertion in five patients, two with the Tuohy and three with the Quincke needle. In one of these cases the stylet had partially passed through the needle tip into the subarachnoid space. In one patient there was difficulty with injection of the first dose, but this was managed by increasing spinal flexion.

The mean maximum height of segmental block after the initial dose of amethocaine was T<sub>8</sub>; the range in the upper level of block was T<sub>4</sub>–L<sub>4</sub> and in the lower level L<sub>5</sub>–S<sub>5</sub>. The block was adequate for surgery 15 minutes after the original bolus of amethocaine in 16 of the 20 patients. In the other four patients there was inadequate cephalad spread at 15 minutes. In one of these the block was very limited, the upper level being S<sub>1</sub> and a general anaesthetic was given. In the other three patients the upper level was between L<sub>1</sub> and L<sub>3</sub> and a small (1.5 ml) dose of hyperbaric bupivacaine 0.5% was sufficient to elevate the block to the required level. There was no evidence of unilateral block in any of the cases and motor block was more than adequate (Bromage 2–3) in all cases.

The average duration of the procedure was 180 minutes and the number of top-ups ranged from 0–4 with an average of 1.33. In one patient a repeat injection was impossible and general anaesthesia was needed to complete surgery. Subsequent investigations showed a kink in the catheter at the point of skin puncture. Surgery became more extensive than planned in one patient and an aortic bifemoral graft was inserted after the upper level of block had been extended by giving a dose of hyperbaric bupivacaine.

A mean of 1.2 (range 0–5) doses of ephedrine 3 mg were required to treat hypotension whenever the decrease in systolic blood pressure was greater than 30%. On direct questioning two patients described low backache postoperatively around the puncture site. Three patients complained of a mild and transient headache and one patient developed a typical postlumbar puncture headache which was treated with an epidural blood patch. A Tuohy needle was used in all of these patients.

### Discussion

In 1940 Lemmon<sup>1</sup> noted that a common problem with spinal anaesthesia using procaine was 'wearing off too soon'. He therefore developed a malleable needle which could be left in place and through which repeated injections could be given. Tuohy improved on this in 1944 by inserting a ureteric catheter. An unacceptably high inci-

dence of postspinal headache associated with the use of the large bore needles, and successful adaption of the continuous method for epidural anaesthesia, resulted in declining use of the continuous spinal technique. The recent development of smaller needles and catheters has prompted renewal of interest.

In our study we chose to evaluate the combination of a 20-gauge needle with a 24-gauge catheter. Smaller sizes are currently available, but after examination it was considered that they would be technically more demanding to handle. Since the evaluation was to be made in elderly patients it was thought that the risk of headache was acceptable.

It is perhaps a little surprising that identification of the subarachnoid space was difficult with a 20-gauge needle in five of 20 patients. This may have been because of unfamiliarity in using the larger needle for lumbar puncture although each anaesthetist used a few catheters before the formal evaluation started. Catheter insertion was more straightforward although a distinct 'knack' had to be acquired when using the Tuohy needle. This is because the stylet must be used to ensure that the catheter advances through the needle; however the catheter will not pass through the curve of the Huber tip with the stylet *in situ*. Therefore catheter advancement and stylet removal had to be coordinated particularly carefully when a Tuohy needle was used.

Our study illustrates a number of advantages associated with the continuous spinal technique. It is possible to titrate the onset as well as the duration of a block so that the desired level is achieved with small increments of local anaesthetic. Figure 1(a) illustrates the way in which an inadequate height of block was quickly corrected with a top-up of bupivacaine, with minimal delay. Although over half of our patients (63%) required increments of ephedrine for hypotension at some point during the procedure, only one required treatment in the first half-hour after the initial dose of the local anaesthetic. The low incidence of hypotension in that period is of particular value in the elderly patient with a background of cardiovascular disease. In common with our experience, Palas<sup>2</sup> found a much lower requirement for vasopressor drugs with continuous spinal anaesthesia than with single shot techniques.

The three major factors influencing the distribution of local anaesthetic in the subarachnoid space are the anatomical level for injection, patient position and the baricity of the local anaesthetic. From experience with epidural catheters it is reasonable to add the direction and the length of the catheter threaded into the space. Figure 1(b) illustrates one case where the L<sub>2-3</sub> space was preferred and the mean and range of the block was shifted upwards. It is of interest that this was a segmental block with sparing of the sacral roots, which in another patient were the only ones blocked (Fig. 1(c)). Figure 2 shows the three possible routes for the catheter to take; caudad, cephalad or to curve around the cauda equina. The first route is a possible explanation for this narrow sacral block. Although we tried to limit the length of catheter lying in the subarachnoid space to 3 cm it is possible that the catheter passed 3 cm in a caudal direction. Added to the flow directed downwards from the single end-holed catheter this might have been sufficient to restrict the height of the block. Whether a lateral-eyed catheter would have helped to increase the distribution of the local anaesthetic is open to debate. The study by Burgess<sup>3</sup> with lateral eyed catheters in an experimental model suggests that it would not.

It is possible that the position which the spinal catheter takes in the subarachnoid space, as well as the flow characteristics of the local anaesthetic emerging from the end hole of the catheter, influences the distribution of the local anaesthetic. Intrathecal injection made through a catheter

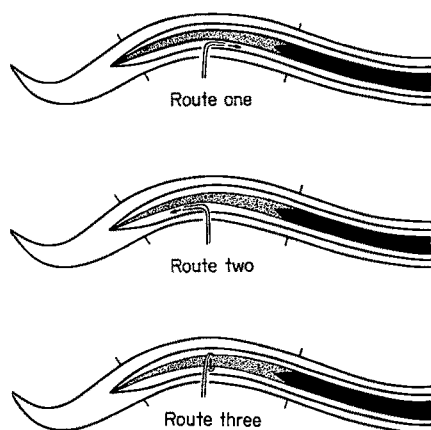


Fig. 2. Three possible routes for spinal catheter: caudad, cephalad or around the cauda equina.

may not spread in the same way as it does after a needle injection and depends on the final position of the catheter tip.

Success of the continuous spinal anaesthetic technique in peripheral vascular surgery will depend on proven benefit over the currently accepted regional anaesthetic technique for these patients, which is epidural block. With familiarity in both techniques there is probably little difference in the

technical difficulty of inserting catheters of 24 gauge or larger. With much finer catheters the technical difficulties are said to be even greater.<sup>4</sup> We would not expect to have to give general anaesthetics to 10% of patients having peripheral vascular surgery under epidural block because of technical difficulties with the catheter, as was the case here. However, the difference may simply be due to familiarity.

The other features of spinal and epidural block are well known and it will require comparative studies to see which technique is to be preferred in the elderly.

#### Acknowledgements

We thank Preferred Medical Products for the gift of spinal catheters for use in this study. Dr L. Morrison was in receipt of a Research Fellowship from ASTRA Pharmaceuticals Ltd.

#### References

1. LEMMON WT. A method for continuous spinal anaesthesia. *Annals of Surgery* 1940; **111**: 141-4.
2. PALAS TAR. Continuous spinal anaesthesia versus single shot technique in the elderly. *Regional Anesthesia* 1989; **14**: s9.
3. BURGESS FW. Influence of the subarachnoid catheter on the distribution of hyperbaric bupivacaine. *Regional Anesthesia* 1990; **15**: s60.
4. HURLEY RJ, LAMBERT DH. Continuous spinal anaesthesia with a microcatheter technique: preliminary experience. *Anesthesia and Analgesia Current Researches* 1990; **70**: 97-102.

*Anaesthesia*, 1991, Volume 46, pages 578-579

### Assessing intravenous cannulation and tracheal intubation training

M. E. Wilson, PhD, FFARCS, Consultant, Department of Anaesthesia, Royal United Hospital, Bath BA1 3NG.

#### Summary

A simple graphical method is described that allows the assessment of a student's progress while learning intravenous cannulation and tracheal intubation. Thirty-four students were studied during their initial training; 91% of those students who obtained a sufficient number of trials to allow a final assessment to be made met the criterion of 80% success for cannulation and 95% achieved similar success in intubation. Thirty-one ambulance staff who returned for a 3-day refresher course were also assessed; 44% (for intravenous cannulation) and 61% (for tracheal intubation) met the 80% success criterion.

#### Key words

*Education*; medical students, paramedical staff.  
*Intubation*; tracheal.  
*Cannulation*; intravenous.

Paramedical staff and medical students are seconded regularly to departments of anaesthesia to learn the skills of intravenous cannulation and tracheal intubation. The following graphical method has been devised to provide an easy assessment of the student's progress.

#### Method

The result of each attempt is recorded sequentially on graph paper by blocking out a small square diagonally upwards for a success and diagonally downwards for a

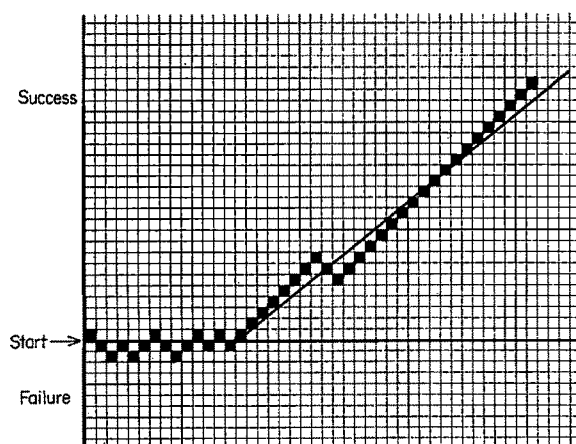


Fig. 1. The performance of a typical student learning intravenous cannulation. The line (drawn at 39° to the x-axis) corresponds to a performance of 80% success.

failure (Fig. 1). Commonly the plot hovers about the abscissa for several attempts as the student struggles to learn the task. Eventually there is a 'Eureka' phenomenon; the task is learnt and most subsequent attempts are successful.

Performance can be assessed quickly by placing a cursor across the graph from the 'Eureka point'. The cursor is a straight line drawn diagonally across a transparent film to represent a predetermined level of performance (e.g. at 39° corresponding to 80% success). This level of performance is achieved if the student's charted zig-zag line of progress crosses and stays above the cursor. Alternatively, the final achievement can be calculated by counting the number of successes in the last 20 trials.

### Results

The graph has been used to assess 12 ambulance staff, 14 Royal Navy Medical Assistants and eight medical students (total 34) during their 2- or 3-week training attachments. All but two of the graphs were of the pattern described or showed immediate evidence of learning, perhaps as a result of transfer of skills from training aids.

For all students, the median number of cannulation attempts was 42 (range 18–103) and the median number of intubation attempts was 27 (range 13–55). Thirty-three students for cannulation and 20 for intubation obtained sufficient opportunities to learn the tasks and then demonstrate their final performance over 20 trials. Their results are shown in Table 1. For cannulation 91% of students, and for intubation 95% of students, achieved the criterion of 80% success.

In addition, the method was used to assess the retention of skills in 31 ambulance staff who had returned for a 3-day refresher period, over a year after initial training. The

Table 1. Successful attempts (percentage of total attempts) at intravenous cannulation and tracheal intubation in the final 20 attempts (after an initial learning period).

	<80%	80–90%	>90%	n
Cannulation	3	22	8	33
Intubation	1	8	11	20

number of attempts obtained was disappointing; the median (range) for cannulation was 16 (9–23) and for intubation 10 (6–17). Eighteen (44%) achieved a calculated success rate of 80% or better for cannulation and 25 (61%) achieved this standard for intubation.

### Discussion

The graph provides a simple and useful means of quantifying achievement. It is popular with students and teachers because they are provided with an immediate picture of the rate and extent of progress. The cursor provides a quick indication of successful training but the number of successes over the last 20 trials provides a more exact measure.

What standard should one expect the trainees to achieve? Although one might wish for a near perfect performance after training, an 80% success rate seems a more realistic aim. Some might consider this too low a standard because potentially difficult patients are seldom presented to trainees. Furthermore, it should be noted that this level of performance may occasionally be achieved during 20 trials by a student whose long-term performance is only 60% successful (with 20 trials, 95% confidence limits of 60% success are 36–81%). Confidence in the estimate would be improved if more trials were examined but this may not be possible within the time available for training. All ambulance staff far exceeded 20 attempts at cannulation and intubation during their initial 3-week attachment, but one of eight medical students, and several of the RN Medical Assistants, failed to obtain this number of attempts at intubation during their shorter attachment. It appears that 2 weeks is scarcely sufficient time to learn and demonstrate the acquisition of these skills and latterly, because of the popularity of the laryngeal mask airway, it has become even more difficult to provide opportunities to intubate the trachea.

Accurate assessment during a 3-day refresher period is impossible with any acceptable statistical confidence. Despite being highly motivated, none of the ambulance staff found 20 opportunities to intubate during this period. The performance of roughly half of the ambulancemen was disappointing one year after training and the 3-day attachment was too brief to provide satisfactory retraining. An annual refresher period of perhaps a week may be required to maintain skills.

## Nausea and vomiting with use of a patient-controlled analgesia system

S. L. Robinson, BA, RGN, RMN, Research Sister, D. Fell, FCAnaes, Senior Lecturer, University Department of Anaesthesia, Leicester Royal Infirmary, Leicester LE1 5WW.

### Summary

*The incidence of nausea and vomiting following patient controlled analgesia and intramuscular morphine injections on demand was compared in a double-blind randomised study of 32 healthy patients undergoing elective cholecystectomy. There were no significant differences between the two groups in mean 24 hour postoperative morphine consumption, subjective experience of pain, nausea and sedation assessed by visual linear analogue scoring, and the postoperative requirements for antiemetic therapy.*

### Key words

*Analgesia; postoperative.*

*Emesis; postoperative.*

*Equipment; patient-controlled analgesia system.*

Patient-controlled analgesia (PCA) systems have been used for the past 20 years as a research tool to develop improved methods of controlling postoperative pain<sup>1</sup> and more recently as an alternative method to administer postoperative analgesia.<sup>2</sup> In clinical use we noted that three patients suffered severe side effects of nausea and vomiting during the use of intravenous morphine by PCA, but did not know if this was caused by the drug or the delivery technique.

The purpose of this study was to compare the incidence of emetic sequelae in patients using PCA devices with a control group of patients receiving regular 4 hourly intramuscular injections of morphine for control of postoperative pain after elective surgery.

### Method

Thirty-two patients scheduled to undergo cholecystectomy gave informed consent to this double-blind randomised controlled study, which was approved by the local Ethics Committee. Patients with a history of psychiatric illness or those receiving opioid therapy pre-operatively were not studied.

Patients were instructed in the use of a Graseby Patient-Controlled Analgesia System (PCAS) at a pre-operative interview. The visual linear analogue scoring (VAS) method that would be used to assess their experience of pain, nausea and sedation was also explained. Each linear analogue comprised a 10-cm unmarked line, the ends of which denoted the extremes of the variables in question, i.e. no pain, worst possible pain; no nausea, worst possible nausea; wide awake, extremely sleepy.

Premedication was with oral diazepam 10 mg given 60–90 minutes before surgery. Anaesthesia was induced with thiopentone via an indwelling intravenous cannula, and tracheal intubation was facilitated by the use of a non-depolarising muscle relaxant. The lungs were ventilated with 67% nitrous oxide in oxygen, supplemented with a volatile agent. Intra-operative opioid was limited to morphine 0.1–0.2 mg/kg. Surgery was performed through a subcostal incision in all cases.

Intravenous morphine was administered in 2-mg incre-

ments in the recovery room at the anaesthetist's discretion until satisfactory analgesia was achieved.

Patients were assigned randomly on return to the ward to one of two groups: group 1 used a PCAS with morphine and received 4 hourly intramuscular injections of saline; group 2 used a PCAS with saline and received 4 hourly intramuscular injections of morphine. Syringes for both the PCAS and injections were prepared by the hospital pharmacy. They were assigned a numerical value and issued in chronological order as each new patient entered the study. The patient's intravenous infusion was connected via a Cardiff valve to the PCAS. The PCAS was programmed to deliver on demand a bolus dose of 1 ml of solution: either 2 mg morphine in 1 ml solution (group 1) or 1 ml of saline (group 2). The lockout interval was set to 10 minutes, thus the maximum hourly patient-controlled dose was 12 mg in group 1. Morphine/saline administration was recorded by a Hewlett-Packard thermal printer attached to the PCAS which recorded both successful and unsuccessful demands.

Morphine or saline were prescribed for all patients to be given intramuscularly 4 hourly: 1 ml (10 mg morphine in group 2) for those who weighed less than 75 kg, or 1.5 ml (15 mg morphine in group 2) for those who weighed more than 75 kg. The ward nurses were instructed to halve or omit these 4 hourly doses if they considered the patient was oversedated or pain-free.

Prochlorperazine (12.5 mg) was prescribed as an antiemetic, to be given only when required and not routinely. Additional prescriptions of morphine 5–10 mg were available as 'escape' analgesia. Linear analogue assessments of pain, nausea and sedation were completed at 2, 4, 6 and 24 hours postoperatively.

Patients completed a short questionnaire at the end of the study period designed to elicit specific information about the occurrence and frequency of vomiting. The questionnaire contained several 'dummy' questions in the attempt to avoid the possibility of bias. (There was a risk that heightened awareness of the possibility of nausea/vomiting might increase patient compliance). The ward nurses, who were unaware of the patient's group, also recorded the incidence of nausea and vomiting over the 24-hour postoperative period.

Table 1. Patient details mean (SD).

	Group 1 PCA morphine	Group 2 intramuscular morphine
<i>n</i>	15	15
Sex; m:f	4:11	4:11
Age; years	60.1 (13.6)	55.3 (16.6)
Weight; kg	66.7 (10.5)	77.7 (10.1)
Duration of surgery; hours	1.5 (0.4)	1.5 (0.6)

Data were analysed by unpaired Student's *t*-test and Wilcoxon rank sum test as appropriate. ANOVA for repeated measures was used for visual linear analogue scores. Chi-squared analyses were applied to administration of antiemetics, episodes of nausea and vomiting and the results of the patients' questionnaires, with Yates' correction as appropriate. The number of patients in the study was chosen to give the study 80% power with a probability of  $< 0.05$  to detect a difference of 20 mm in VAS for nausea, the variable at issue. We deemed this 20% difference to be clinically important.

### Results

All patients were Caucasian, aged 26–78 years and ASA grades 1 and 2. Two patients were excluded because they received the other's injection in error, despite clear labelling. Data were thus available for 30 patients (15 in each group) who were well matched for age, gender and duration of operation but group 2 patients were significantly heavier than group 1 ( $p < 0.05$ ) (Table 1). There were no significant differences in the quantities of morphine used in the operating theatre, recovery room or in the 24-hour postoperative consumption between the two groups (Table 2).

Three patients required 'escape' analgesia. One patient in group 1 needed two doses of 5 mg morphine and two patients in group 2 required two doses of 10 mg morphine. All patients continued to use the PCAS and completed the study. Three patients in group 2 (saline), who used the PCAS, were sufficiently pain-free to refuse the routine injections and therefore did not receive any postoperative analgesia. There were no significant differences between the two groups in the overall requirements of antiemetics or in the frequency of nausea and vomiting as recorded by the ward nurse. Eight patients in group 1 did not vomit and did not require antiemetics compared with four patients in group 2 (Table 3).

Linear analogue scores for pain, nausea and sedation were not statistically different between the groups (Table 4). The results of the patients' questionnaires were not statistically significant and corresponded with the ward nurses' assessments of nausea and vomiting.

Table 2. Morphine consumption in the first 24 hours (mg); mean (SD).

	Group 1 PCA morphine	Group 2 intramuscular morphine
Intra-operative	11.4 (4.9)	12.1 (5.0)
Recovery	3.4 (4.6)	4.2 (3.2)
Post-operative	34.8 (19.3)	30.2 (26.0)
Total	49.6 (21.3)	46.6 (27.1)

Table 3. Number of patients who were nauseated, vomited and received antiemetics.

	Group 1 PCA morphine	Group 2 intramuscular morphine
Nauseated	4	7
Vomited	4	10
Received prochlorperazine	7	10
Not nauseated did not vomit did not receive prochlorperazine	8	4

### Discussion

We have demonstrated in this study that morphine administered with a PCAS causes no more nausea and vomiting than the conventional intramuscular method.

We found a statistically significant difference in weight between the two groups, but others have not found weight to correlate with opioid usage from a PCAS<sup>3</sup> and, since all patients titrated their needs to an acceptable level of comfort, we do not feel that this variable affects the conclusions drawn from this study.

There was a total of 20 episodes of vomiting in the active intramuscular group; the patients required 20 doses of antiemetic compared with 11 and 8 (respectively) in the active PCA group. Four patients in the PCA group vomited compared with 10 in the intramuscular group, whilst eight patients in the PCA group did not vomit and were not nauseated compared with four patients in group 2. This trend may indicate a favourable aspect of opioid analgesia administered via PCAS. The possibility of a Type II error was minimised in this study by selecting a difference of 20 mm in respect of nausea and vomiting, which, together with the number of patients in each group and an estimate of the standard deviation for nausea of 20 mm found from previous work, gives the study a power of approximately 80%.<sup>5</sup>

Pain and sedation visual analogue scores between the groups did not vary significantly and it is interesting to note that three patients in group 2 did not receive any postoperative analgesia yet all had triggered the PCA device. This confirms the work of others<sup>4</sup> which has shown the

Table 4. Visual linear analogue scores (mm); mean (SD)

	Hours	Group 1 PCA morphine	Group 2 intramuscular morphine
Pain	2	2.3 (9.0)	3.1 (12.1)
	4	12.3 (22.6)	7.5 (25.0)
	6	15.3 (22.1)	20.0 (35.1)
	24	26.3 (13.1)	21.1 (15.5)
Nausea	2	0 (0)	4.7 (15.1)
	4	0.8 (2.6)	0.9 (3.6)
	6	11.3 (28.1)	18.4 (34.4)
	24	17.5 (29.8)	15.1 (24.5)
Sedation	2	98.3 (6.4)	92.6 (19.5)
	4	81.9 (30.9)	96.5 (13.4)
	6	76.7 (35.6)	82.3 (33.0)
	24	66.0 (26.0)	55.5 (34.2)

wide variation in postoperative analgesic requirements. It also suggests a placebo effect of PCA. It is our observation that patients are more relaxed before surgery with the knowledge that they are to be in control<sup>6</sup> and will not have to wait for a nurse to administer analgesia. This is particularly true of patients who have previously undergone surgery.

Welchew and colleagues<sup>7</sup> assessed the emetic sequelae in a study which compared intramuscular analgesia with PCA. However, although nausea was found to be significantly less frequent in patients in the regular intramuscular morphine group than with fentanyl administered by PCA, the use of two different opioids may invalidate this finding. In addition, if analgesia is given via the intramuscular route on a regular basis it may be as satisfactory as PCA but this rarely occurs in a ward. We considered it to be unethical to compare PCAS with 'on demand' morphine because this would have necessitated giving placebo 'on demand' for pain, which clearly is unacceptable. Instead we asked the ward nurses to omit or reduce the injections if the patient refused analgesia or if they thought that the patient was oversedated; thus customary practice was not followed precisely in our study.

Owen and colleagues<sup>8</sup> examined three different doses (0.5, 1 and 2-mg boluses) of morphine administered via PCA in a range of postoperative conditions and, whilst he found that the optimum dose for analgesia was 1 mg, he did not find any difference in the incidences of nausea, vomiting and administration of antiemetics between the groups.

We conclude that the use of morphine administered via a PCAS for postoperative analgesia causes no more nausea

and vomiting than conventional injections. The results from this study suggest that fewer patients who self administer morphine may require antiemetic injections compared with those who receive intramuscular morphine therapy.

## References

1. SECHZER PH. Studies in pain with the analgesic-demand system. *Anesthesia and Analgesia* 1971; **50**: 1-10.
2. NOTCUTT WG, MORGAN RJM. Introducing patient-controlled analgesia for postoperative pain control into a district general hospital. *Anaesthesia* 1990; **45**: 401-6.
3. TAMSEN A, HARTVIG P, DAHLSTRÖM B, LINDSTRÖM B, HILSON HOLMDAHL M. Patient controlled analgesic therapy in the early postoperative period. *Acta Anaesthesiologica Scandinavica* 1979; **23**: 462-70.
4. DAHLSTRÖM B, TAMSEN A, PAALZOW L, HARTVIG P. Patient controlled analgesic therapy, part 4: pharmacokinetics and analgesic plasma concentrations of morphine. *Clinical Pharmacokinetics* 1982; **7**: 266-79.
5. ALTMAN DG. Statistics and ethics in medical research. 3 How large a sample? *British Medical Journal* 1980; **281**: 1336-8.
6. EGAN KJ. What does it mean to a patient to be "in Control"? In: FERRANTE FM, OSTHEIMER GW, COVINO BG, eds. *Patient-controlled analgesia*. Cambridge, Massachusetts: Blackwell Scientific Publications, 1990; 17-27.
7. WELCHEW EA. On-demand analgesia. A double-blind comparison of on-demand intravenous fentanyl with regular intramuscular morphine. *Anaesthesia* 1983; **38**: 19-25.
8. OWEN H, PLUMMER JL, ARMSTRONG I, MATHER LE, COUSINS MJ. Variables of patient-controlled analgesia 1. bolus size. *Anaesthesia* 1989; **44**: 7-10.

*Anaesthesia*, 1991, Volume 46, pages 582-585

## Incidence and management of airway problems in the CHARGE Association

C.G. Stack, BSc, MBBS, FFARCS, Registrar, R.K.H. Wyse, MRCP, Senior Lecturer, Departments of Anaesthetics and Paediatric Cardiology, The Hospitals for Sick Children, Great Ormond Street, London WC1 3JH.

## Summary

*The airway problems associated with anaesthesia in patients with the CHARGE Association have been underreported. We undertook a retrospective review of 50 cases, of which anaesthetic records were available for 37 patients. Apart from choanal atresia and cleft lip and palate, 56% of patients had some other upper airway abnormality. There appeared to be greater difficulty in tracheal intubation with increase in age in four patients. The incidence and management of airway abnormalities are discussed.*

## Key words

*Complications; airway, tracheal intubation. Syndrome; CHARGE Association.*

In 1979, Hall described 17 patients with multiple congenital abnormalities associated with choanal atresia which did not fit into any previously described syndrome.<sup>1</sup> In 1981, Pagon *et al.* described 21 similar patients and defined these

anomalies as the CHARGE Association.<sup>2</sup> The major anomalies are: C, coloboma of the eye; H, heart disease; A, atresia of the choanae; R, retarded growth, development and/or central nervous system anomalies; G, genital hypo-

**Table 1.** Incidences of main diagnostic features of CHARGE Association found in 50 patients.

Feature	n
Coloboma	44
Heart disease	45
Choanal atresia/stenosis	28
Retardation—growth	43
—mental	39
Genital abnormalities (male)	28/29
Ear abnormalities	50

**Definitions**

Heart disease: majority conotruncal or patent ductus arteriosus.

Growth retardation: <3rd centile.

Mental retardation: >2 SD below mean.

Genital: undescended testis or micropenis.

Ear: deafness and/or external ear deformity.

plasia in males; E, ear anomalies and/or deafness. Davenport, Hefner and Mitchell consider that at least four of these major criteria have to be present for the diagnosis of CHARGE Association to be made.<sup>3</sup>

Other features of varying severity have been reported, including facial palsy, renal anomalies, tracheo-oesophageal fistula, feeding difficulties, skeletal abnormalities, cleft lip and palate and micrognathia.<sup>4</sup>

Personal experience with airway problems in patients with the CHARGE Association, led us to believe that the incidence and variety of airway abnormalities were higher than previously reported. Consequently, we decided to review all cases referred to The Hospitals for Sick Children during the last 10 years. This review discusses the different airway problems, their incidence and anaesthetic management. A separate review of these patients' medical problems and management has been published.<sup>5</sup>

**Methods**

We conducted a retrospective search through the hospital coding system, genetic departmental records at the Institute of Child Health and through the cardiological departmental records of patients with multiple congenital anomalies. This search was based on a previously reported study of the association in 20 patients.<sup>6</sup>

Patients were diagnosed as having CHARGE Association if they had at least four of the major criteria, as described by Davenport, Hefner and Mitchell.<sup>3</sup> Patients who had incomplete data were excluded from the series, even if they satisfied this definition. Patients with only three of the major criteria were considered to form a 'near miss' group.

All records of patients who satisfied the above diagnosis of CHARGE Association and who had been admitted between 1 April 1979 and 1 April 1989 were reviewed. Specific problems at the time of anaesthesia or in the intensive care unit, and any airway problems recorded by the ENT surgeons at laryngoscopy or bronchoscopy, were noted.

**Results**

Fifty-six patients were considered to have probable CHARGE Association. Of these, two did not fit the criteria for inclusion fully and thus could be considered near miss CHARGE Association. Four patients, although satisfying the criteria, had insufficient data for full analysis and were therefore excluded. Thus 50 patients satisfied the criteria for CHARGE Association and were included in this study.

Twenty-nine patients were male, and 21 were female.

**Table 2.** Frequencies of other reported features of CHARGE Association found in 50 patients.

Feature	n
Facial nerve palsy	22
Renal anomalies	12
Tracheo-oesophageal fistula	7
Gastro-oesophageal reflux	25
Cleft lip	5
Cleft palate	5
Short neck	17
Micrognathia	20
Hypotonia	23

Thirteen had died between the ages of 8 days and 17 years, although most deaths occurred within the first year of life. The age range of survivors was 4 months to 26 years at the time of this review.

Tables 1 and 2 show the incidences of the main diagnostic and other associated features in this series. Two important associated features to note are the high frequencies of gastro-oesophageal reflux (50%) and muscular hypotonia (46%).

Thirty-seven patients had undergone general anaesthesia at this hospital. A further 10 patients underwent general anaesthesia at other hospitals, from which records were not obtained. Only three patients had never been anaesthetised at the time of this review.

Table 3 shows the incidences of upper airway abnormalities in the patients.

**Choanal atresia.** Choanal atresia or stenosis was present in 56% of patients (Table 1); the incidences of the different types are shown in Table 3. Twenty-one patients required operative correction of the airway obstruction. Fifteen of these required redilatation, and three of these needed re-operation.

**Micrognathia.** Twenty patients had documented micrognathia. Sixteen were anaesthetised at the Hospitals for Sick Children; 11 had no recorded problems related to anaesthesia or tracheal intubation. Three patients with no recorded intubation problems had a tracheostomy, two to aid weaning following cardiac surgery and one because of severe laryngomalacia.

Five patients with micrognathia had problems at intubation. Three of these patients were found to have a very anterior larynx, with prominent micrognathia at the time of direct laryngoscopy. This led to difficult intubation as the vocal cords could not be seen at laryngoscopy. A bougie was necessary to aid placement of the tracheal tube. Tracheal intubation appeared to be more difficult with increasing age in all three of these patients (aged 15, 34 and 42 months at the time of the most recent intubation). A fourth patient, with an anterior larynx, was difficult to

**Table 3.** Incidences of upper airway abnormalities in CHARGE Association found in 50 patients.

Feature	n
Choanal atresia bilateral	16
unilateral	4
stenosis bilateral	3
unilateral	5
Micrognathia	20
Laryngomalacia	4
Subglottic stenosis	3
Bulbar palsy	1
Laryngeal cleft	1
Laryngeal web	1
Recurrent laryngeal nerve palsy	1



intubate, but intubation was performed after lateral pressure had been applied to the larynx. A fifth patient with micrognathia, hypotonia and pharyngeal incoordination underwent cleft palate repair at 6 months of age. He developed postoperative stridor due to laryngeal oedema after intubation with a snug-fitting 3.5-mm uncuffed oral tracheal tube, which is smaller than expected for his age. The stridor settled after re-intubation, elective overnight ventilation and intravenous dexamethasone 0.1 mg/kg 6-hourly and his trachea was extubated uneventfully. Subsequently, at the age of 9 months, intubation was found to be more difficult, requiring the use of a straight-bladed laryngoscope with cricoid pressure in order to lift the epiglottis sufficiently to obtain a view of the vocal cords. There was no obvious connexion between these two events.

**Laryngomalacia.** Four patients were found to have laryngomalacia at direct laryngoscopy under general anaesthesia. Two main problems were evident. Firstly, during induction, maintenance of the airway was particularly difficult. The use of continuous positive airways pressure (CPAP) (by partial occlusion of the bag at the end of the T-piece) helped to rectify this situation by splinting the floppy larynx more open. However, one child needed to be turned on to his side to maintain an adequate airway during induction.

Two of these patients were able to maintain an airway only with long-term intubation or CPAP via a nasal prong. Tracheostomy was required for long-term airway management in both children.

**Subglottic stenosis.** Three patients were found to have subglottic stenosis at direct laryngoscopy. Two had previously required tracheal intubation at birth for respiratory distress, each with a 3.0 mm uncuffed tracheal tube. One of these patients had developed stridor by the age of 4 months. Subglottic stenosis was confirmed, and caused difficulty with tracheal intubation on a number of occasions. A 3.0 mm uncuffed Portex tracheal tube could be passed when he was 2 years old. By the age of 8 years, the stridor had disappeared and his trachea was intubated with a 5.0 mm uncuffed tube (still small for his age). The second patient required a Blalock-Taussig shunt for Fallot's tetralogy on the first day of life. She was intubated with a 3.0 mm uncuffed tracheal tube. Her trachea could not be extubated and by 10 days of age because of subglottic stenosis, tracheostomy was performed. The third patient was not intubated until the age of seven months. She had inspiratory stridor, especially during feeding. Examination under anaesthesia showed unilateral choanal atresia and mild subglottic stenosis. A 3.5 mm uncuffed tracheal tube was the largest size that could be inserted.

**Miscellaneous.** One infant has a first-degree laryngeal cleft with an incompetent larynx. He had pharyngeal incoordination with considerable problems of gastro-oesophageal reflux. Intubation was not difficult as long as the tracheal tube was long enough to pass beyond the cleft. One infant had a congenital laryngeal web which necessitated tracheostomy at 2 weeks of age. He subsequently required laryngotracheoplasty to allow decannulation. However, his right vocal cord became fixed and was replaced by fibrous tissue. One infant with profound hypotonia and laryngomalacia also had an idiopathic left recurrent nerve palsy. She underwent three operations which were all complicated by postoperative chest infections that required CPAP and physiotherapy. One infant with gastro-oesophageal reflux and recurrent aspiration was diagnosed as having bulbar palsy. He underwent tracheostomy at the age of 6 weeks.

**Tracheostomy.** A tracheostomy was required in seven patients. Two were performed in postoperative cardiac patients to aid long-term ventilation and weaning. The

other five were related to problems with the upper airway: two because of laryngomalacia; one for a laryngeal web; one for subglottic stenosis; and one for bulbar palsy.

**'Near miss' CHARGE.** Two patients with only three of the main features of CHARGE Association were excluded from the main review. One had Fallot's tetralogy, abnormally shaped ears with deafness and growth retardation. He also had a tracheo-oesophageal fistula and micrognathia. He underwent surgery on day one for repair of the fistula. Tracheal intubation was relatively easy. However, intubation at 7 months of age was very difficult because of micrognathia and the anterior position of his larynx. The second patient did not have any airway problems.

### Discussion

Graham suggested that the findings in CHARGE Association can, to a certain extent, be explained by arrested development *in utero* between the 35th and 45th days of gestation. The defects appear to be predominantly midline in type.<sup>4</sup> The causes are unknown but there is a small familial incidence of approximately 1%, suggesting heterogeneous causes in the remainder.

CT scan evidence suggests that the choanal atresia which occurs in this association may lead to a narrower posterior choanal region with a more contracted nasopharynx than in patients who have isolated choanal atresia.<sup>7</sup> The re-operation rate was found to be much higher in the CHARGE Association group.

There are three common, but little recognised, airway features of CHARGE Association. In patients with micrognathia, our experience shows that intubation may be difficult due to the combination of a small jaw and anterior larynx. Intubation may continue to be difficult with increasing age. Laryngomalacia may lead to upper airway collapse during light anaesthesia and require CPAP during induction of anaesthesia. Tracheostomy may be needed. Subglottic stenosis was probably acquired in two patients in this series but congenital in the third. In all cases the airway increased in size with age.

Careful pre-operative assessment is required in patients with multiple congenital abnormalities, particularly if airway and cardiac defects are present. Anaesthesia may be complicated by other problems of the association such as cardiac lesions, gastro-oesophageal reflux and muscular hypotonia. There have been no reports of problems associated with neuromuscular blocking drugs in patients with muscular hypotonia, although in this series there were problems with weaning and extubation.

We recommend that the anaesthetic technique for these patients should include atropine (0.02 mg/kg) for premedication, followed by gaseous induction with 100% oxygen and halothane. Tracheal intubation under deep halothane anaesthesia is preferable. This technique can be modified to take account of the features of individual patients. In our experience, patients with micrognathia should receive a gaseous induction, even if tracheal intubation has been successful previously because of the possible risk of increasingly difficult intubation. Awake intubation is an alternative in the neonate.

It is essential to have a range of appropriate tracheal tubes, intubating equipment such as bougies and a range of different laryngoscopes readily available. Equipment for cricothyroid puncture or perhaps, in older children, mini-tracheostomy should also be available. Tracheostomy may be necessary for long-term airway management.

Airway problems are common in the CHARGE Association. Micrognathia in combination with an anterior larynx may lead to difficulty in intubation of the trachea.

Patients with near miss CHARGE Association may also offer unexpected airway problems.

### Acknowledgments

We thank Dr. D.J. Hatch and Dr E. Battersby for their helpful comments and suggestions on the manuscript.

### References

1. HALL BD. Choanal atresia and associated multiple anomalies. *Journal of Pediatrics* 1979; **95**: 395-8.
2. PAGON RA, GRAHAM JM JR, ZONANA J, YONG S-L. Coloboma, congenital heart disease, and choanal atresia with multiple anomalies: CHARGE association. *Journal of Pediatrics* 1981; **99**: 223-7.
3. DAVENPORT SLH, HEFNER MA, MITCHELL JA. The spectrum of clinical features in CHARGE syndrome. *Clinical Genetics* 1986; **29**: 298-310.
4. GRAHAM JM JR. CHARGE Association. In: SMITH DW, ed. *Recognizable patterns of malformation: major problems in clinical pediatrics*, 3rd edn. Philadelphia; Saunders, 1982: 522-4.
5. BLAKE K, RUSSELL-EGGITT IM, MORGAN DW, RATCLIFFE JM, WYSE RKH. Who's in CHARGE? Multidisciplinary management of patients with CHARGE association. *Archives of Disease in Childhood* 1990; **65**: 217-23.
6. OLEY CA, BARAITSER M, GRANT DB. A reappraisal of the CHARGE association. *Journal of Medical Genetics* 1988; **25**: 143-56.
7. CONGILIO JU, MANZIONE JV, HENGERER AS. Anatomic findings and management of choanal atresia and the CHARGE association. *Annals of Otology, Rhinology and Laryngology* 1988; **97**: 448-53.

*Anaesthesia*, 1991, Volume 46, pages 585-587

### Extradural vein puncture—an avoidable complication

D. Mannion,\* MB, BCh, BAO, FFARCSI, Registrar, R. Walker, MB, ChB, FFARCS, Senior Registrar, K. Clayton, MB, ChB, FFARCS, Consultant, Walsgrave Hospital, Clifford Bridge Road, Walsgrave, Coventry CV2 2DX.

### Summary

*Accidental cannulation of an extradural vein is a troublesome and potentially serious complication of extradural catheter insertion. This study was conducted to assess the influence of posture, catheter size and the injection of saline before catheter insertion, on its occurrence. Eight different techniques were studied based on combinations of these three factors. There was no difference in incidence with respect to posture. The use of 18-gauge catheters, after injection of 10 ml of 0.9% saline, resulted in a significant ( $p < 0.01$ ) reduction in the incidence of extradural vein cannulation. This technique is recommended in obstetric patients as a means of avoiding accidental intravenous injection of local analgesic.*

### Key words

*Anaesthesia; obstetric.  
Anaesthetic techniques, regional; epidural.  
Complications; vessel puncture.*

Extradural analgesia is the most successful method of pain relief in labour and is associated with a low incidence of serious complications. There are, however, a number of potential complications which may be life-threatening, if undetected before administration of local analgesic.

One such complication is the inadvertent cannulation of an extradural vein, the incidence of which, in obstetric practice, varies between 1%<sup>1</sup> and 10%.<sup>2,3</sup> This may result in systemic toxicity of local analgesic,<sup>4</sup> inadequate pain relief<sup>5</sup> or extradural haematoma formation with neurological complications.<sup>6</sup> It is uncertain which factors influence the occurrence of extradural vein cannulation. There is evidence that the incidence is reduced by injection of 10 ml of bupivacaine into the extradural space, before catheter insertion.<sup>7</sup> McNeill and Thorburn showed no difference in incidence when 16- or 18-gauge catheters are used.<sup>8</sup> It

occurs more frequently in parturients, presumably because of distension of extradural veins.<sup>9</sup> It is possible therefore that variations in venous distension associated with different postures during catheter insertion could affect the incidence.

This study was carried out to assess the combined influence of patient posture, catheter size and infusion of 0.9% saline on the incidence of venous puncture by the extradural catheter.

### Method

The study was carried out on 217 patients in established labour who requested extradural analgesia. Their ages ranged from 16 to 40 years. Informed consent was obtained

\* Present address: Anaesthetic Tutor, Department of Anaesthesia, Meath Hospital, Heytesbury Street, Dublin 8, Ireland.  
Accepted 9 November 1990.

Table 1. Study groups.

Group	Catheter gauge	Position	Saline?
1	18	Sitting	Saline
2	18	Sitting	No saline
3	18	Lying	Saline
4	18	Lying	No saline
5	16	Lying	Saline
6	16	Lying	No saline
7	16	Sitting	Saline
8	16	Sitting	No saline

from each patient after a full explanation of the proposed technique.

Each patient had a 16-gauge intravenous cannula inserted and their circulations were preloaded with 1000 ml of compound sodium lactate solution. Blood pressure measurements were made using a noninvasive monitor (Critikon Dinamap) before the procedure and at 5-minute intervals thereafter for 30 minutes.

Eight different techniques were studied based on the position of the patient during insertion, the size of catheter used and the injection or otherwise of 10 ml of 0.9% saline into the extradural space before catheter insertion (Table 1).

The extradural space was located with a Tuohy needle at L<sub>2-3</sub> or L<sub>3-4</sub> intervertebral spaces using the loss of resistance to air technique. Those receiving saline then had 10 ml of saline injected via the Tuohy needle, between uterine contractions. An 18-gauge or 16-gauge Portex multi-orifice catheter (reference number 100/394/116 or 100/394/118) was threaded through the needle between contractions, until the 15-cm mark was at the hub. The Tuohy needle was withdrawn over the catheter, which was then positioned so as to leave 3 cm in the extradural space.

The catheter was then aspirated and the presence of any blood or blood stained fluid within the lumen was taken as a positive result, i.e. that venous puncture had occurred. If this happened the catheter was withdrawn until no further blood was aspirated. If this manoeuvre was unsuccessful, the catheter was resited in another space. In cases in which the catheter needed to be resited, the result of the first insertion only was included in the study.

Once the extradural catheter was correctly positioned the patient was given a test dose of bupivacaine 3 ml 0.5%. Five minutes later a top-up dose of bupivacaine 7 ml 0.5% was given. After 30 minutes the height of sensory blockade was assessed using ethyl chloride spray.

The results were analysed using analysis of variance, Chi-squared test and a three-way test of partition using the G-statistic. A probability value of less than 5% was considered significant.

### Results

There was no significant difference between the groups with respect to age, weight, parity, cervical dilatation at the time of catheter insertion, or depth of the extradural space from skin. The height of block achieved was similar in all groups whether or not saline was injected before the local analgesic.

The number of patients in each group in whom blood was aspirated is shown in Table 2. There was no significant difference in the incidence between the lying (11 patients) and sitting positions (15 patients).

The effect of the operator's technique of insertion was then examined. Table 3 shows the results based on the size of Tuohy needle used and the use, or otherwise, of saline,

Table 2. Incidence of aspiration of blood from the extradural catheters in the eight groups of patients.

Group	n	Blood aspiration (n)	Blood aspiration (% of group)
18-G/ Sitting/saline	28	0	0%
18-G/ Sitting/no saline	25	4	16%
18-G/ Lying/saline	30	0	0%
18-G/ Lying/no saline	27	5	19%
16-G/ Lying/saline	27	4	15%
16-G/ Lying/no saline	28	2	7%
16-G/ Sitting/saline	26	3	12%
16-G/ Sitting/no saline	26	8	31%

independent of the effect of posture. It can be seen that the technique using an 18-gauge Tuohy needle and 10 ml of saline is significantly better ( $p < 0.01$ ) than the three other techniques.

### Discussion

Previous studies have reported the incidence of aspiration of blood to be between 1 and 10%. In our study there was an overall incidence of 12%. However, this varied between the different groups from 0–31%. The use of 18-gauge cannulae and 10 ml of saline before insertion of the extradural catheter was clearly shown to be superior to any other technique, irrespective of posture during insertion.

The injection of saline distends the extradural space, decreasing the likelihood of venous cannulation. This technique was first suggested by Bromage<sup>1</sup> and its efficacy was confirmed by Verniquet using 0.5% bupivacaine.<sup>7</sup> The results of our study suggest that it is the mechanical distension that is effective rather than any pharmacological effect of bupivacaine.

The superiority of 18-gauge catheters has not previously been documented. McNeill and Thorburn failed to demonstrate any difference between 18- and 16-gauge catheters in terms of vessel puncture.<sup>8</sup> Our study suggests, however, that, when used with saline, 18-gauge catheters are superior. It is not clear why the combination of a 16-gauge catheter and saline did not confer benefit. It is possibly because the 18-gauge catheters are softer. The use of softer extradural catheters has been shown to result in less dural or blood vessel perforations.<sup>10</sup>

The importance of avoiding cannulation of the extradural veins is obvious. If recognised, it is a troublesome complication. If unrecognised, the consequences may be serious. Since extradural analgesia became popular in 1968 nine deaths associated with its use have been reported by the Confidential Enquiries into Maternal Deaths in England and Wales. In 1985, in a review of 27 000 obstetric extradural anaesthetics, Crawford reported nine potentially life-threatening complications, one third of which were associated with accidental intravenous injection of local

Table 3. Incidence of aspiration of blood from the extradural catheter in four groups of patients; effects of catheter size (18-gauge or 16-gauge) and injection of saline before catheter insertion.

Group	n	Blood aspiration (n)	Blood aspiration (% of group)
18-G, saline	58	0	0%
18-G, no saline	52	9	17%
16-G, saline	53	7	13%
16-G, no saline	54	10	19%

analgesic.<sup>11</sup> The inadequacy of conventional tests to detect cannulation of a vein when multiorifice catheters are used, has been demonstrated; the aspiration test failed in 70% of cases.<sup>10</sup> If the incidence of complications from extradural analgesia is to be reduced, then avoidance of vessel puncture by the catheter is of considerable importance. We suggest that this can be achieved in obstetric patients by the use of an 18-gauge catheter, inserted after injection of 10 ml saline into the extradural space.

#### Acknowledgments

We are grateful to Mr M. Keating, Trinity College Dublin, for assistance with statistical analysis.

#### References

1. BROMAGE PR. *Epidural analgesia*. Philadelphia: W.B. Saunders, 1978.
2. MOIR DD, THORBURN J. *Obstetric anaesthesia and analgesia*, 3rd edn. London: Bailliere Tindall, 1986.
3. CRAWFORD JS. *Principles and practice of obstetric anaesthesia*, 4th edn. Oxford: Blackwell Scientific Publications, 1978.
4. RYAN DW. Accidental intravenous injection of bupivacaine: a complication of obstetrical epidural anaesthesia. *British Journal of Anaesthesia* 1973; **45**: 907-8.
5. HYLTON RR, EGER EI, ROVNO SH. Intravascular placement of epidural catheters. *Anesthesia and Analgesia* 1964; **43**: 379-82.
6. USUBIAGA JE. Neurological complications following epidural anaesthesia. *International Anesthesiology Clinics* 1975; **13**: 1-153.
7. VERNIQUET AJW. Vessel puncture with epidural catheters. Experience in obstetric patients. *Anaesthesia* 1980; **35**: 660-2.
8. MCNEILL MJ, THORBURN J. Cannulation of the epidural space. A comparison of 18- and 16-gauge needles. *Anaesthesia* 1988; **43**: 154-5.
9. GALBERT MW, MARX GF. Extradural pressures in the parturient patient. *Anesthesiology* 1974; **40**: 499-502.
10. BECK H, BRASSOW F, DOHEN M, BAUSE H, DZIADZKA A, SCHULTE AM ESCH J. Epidural catheters of the multi-orifice type: dangers and complications. *Acta Anaesthesiologica Scandinavica* 1986; **30**: 549-55.
11. CRAWFORD JS. Some maternal complications of epidural analgesia for labour. *Anaesthesia* 1985; **40**: 1219-25.

## Correspondence

<b>Attenuation of suxamethonium myalgias</b>	588	<b>Ethyl chloride and diethyl ether for assessing the extent of epidural blockade</b>	596
<i>A.S. Laurence, FFARCS and S.O. Smith</i>		<i>W.A. Lindsay, FFARCS</i>	
<i>M.L. Mingus, MD, A. Herlich, MD and J.B. Eisenkraft, MRCP, FFARCS</i>	589	<b>Assessment of training in anaesthesia and related skills</b>	597
<b>Trauma to the posterior pharyngeal wall caused by a laryngeal mask airway</b>	589	<i>P.G. Lawler, FRCP, FFARCS, V.R. Patla, MD, E. Garcia, LSM and N. Puttick, FFARCS</i>	
<i>R. Marjot, FCAnaes</i>		<b>Block of epidural needle by blood clots</b>	597
<b>Laryngeal mask airway in children: insertion technique</b>	590	<i>E.T.M. Lim, MMed(Anaes), T.S. Teh, MB, BS and Y.H. Li, MB, BS</i>	
<i>B.F.M. Chow, FCAnaes, M. Lewis, FFARCS and S.E.F. Jones, FFARCS</i>		<b>Ketamine, midazolam and vecuronium infusion</b>	598
<b>Laryngeal masks and chair dental anaesthesia</b>	591	<i>P.J. Ward, DA and J. Restall, FFARCS</i>	
<i>H. Noble, FCAnaes and D.J. Wooller, MB, BS</i>		<b>Blood clot obstruction of the upper airway</b>	598
<b>Yet another use for the laryngeal mask airway</b>	591	<i>I.A. Ewart, FFARCS and M. Weston, FFARCS</i>	
<i>R.W.M. Walker, FCAnaes and D. Murrell, FFARCS</i>		<b>Capnography and sticking valves in the circle system</b>	599
<b>Unexpected position of the laryngeal mask airway</b>	592	<i>F.A. Rosewarne, FFARCS</i>	
<i>A.R. Molloy, FFARCS</i>		<b>32-gauge spinal catheters through 26-gauge spinal needles</b>	599
<b>Aspiration pneumonia and the laryngeal mask airway</b>	592	<i>N.M. Denny, FFARCS</i>	
<i>A.W. Pearce, FCAnaes and M.L. Heath, FCAnaes</i>		<b>Fixation of nasotracheal and nasogastric tubes</b>	600
<b>Malfunctioning of disposable breathing systems</b>	592	<i>H. Middleton, FFARCS</i>	
<i>S. Shorey, MD and M. Ravishankar, MD</i>		<b>Pulmonary artery catheter entrapment</b>	600
<i>S.K. Williams</i>	593	<i>C. Lanigan, FCAnaes and E. Cornwell, FFARCS</i>	
<b>Pneumatic tourniquets</b>	593	<b>Central venous cannulation: a complication</b>	601
<i>M.L. Heath, FCAnaes</i>		<i>C.P.J. Morton, FCAnaes and N.M. Dearden, FFARCS</i>	
<b>Cuff failure—a complication of tracheal intubation</b>	593	<i>J.P. Wilkinson</i>	602
<i>R. Williamson, FFARCS and A.M. Gorven, FFA(SA)</i>		<b>Freezing thiopentone solution</b>	602
<b>Precordial stethoscopes</b>	594	<i>M.H. Cross, DA</i>	
<i>A. Kumar, MD, V.K. Grover, MD, I. Bhukal, MD and S. Sharma, MD</i>		<b>The anaesthetic management of tracheal stenosis</b>	602
<b>Guide wire embolism</b>	595	<i>I.G. Grove-White, FFARCS</i>	
<i>P. Barker, FCAnaes</i>		<b>Contiplex (Braun) Katheterset</b>	603
<b>Upper airway obstruction by enlarged tonsils</b>	595	<i>G.A. Fritz, MD and E.G. Bradshaw, FFARCS</i>	
<i>W. Konarzewski, FFARCS, P. Walker, MB, BS and A. Donovan, DA</i>		<i>H.J. Otto</i>	603
<b>Is the pre-operative removal of dentures necessary?</b>	596		
<i>M. Coble, FFARCS and J. Dunne, FFARCS</i>			
<b>Coagulation screening before epidural analgesia in pre-eclampsia</b>	596		
<i>T.N. Trotter, FCAnaes, A.L. Armstrong, FIMLS, J.K. Wood, FRCPath and A.E. May, FCAnaes</i>			

### Attenuation of suxamethonium myalgias

We were interested to read the paper by Mingus, Herlich and Eisenkraft (*Anaesthesia* 1990; 45: 834–7). The failure of the authors to demonstrate a worthwhile reduction of postoperative suxamethonium myalgia in their daycase laparoscopy patients is perhaps because of the method of assessment they used (telephone enquiry). No mention of

postoperative analgesia was given in the paper, especially since presumably the patients would have been administering their own medication at home. Assessment of postoperative analgesia by telephone enquiry must make it difficult to distinguish pain caused by suxamethonium from pain caused by the operative procedure itself. Just because

All correspondence should be addressed to Dr M. Morgan, Editor of *Anaesthesia*, Department of Anaesthetics, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0HS, United Kingdom.

Letters must be typewritten on one side of the paper only and double spaced with wide margins. Copy should be prepared in the usual style and format of the Correspondence section. Authors must follow the advice about references and other matters contained in the Notice to Contributors to *Anaesthesia* printed at the back of each issue. The degrees and diplomas of each author must be given in a covering letter personally signed by all the authors.

Correspondence presented in any other style or format may be the subject of considerable delay and may be returned to the author for revision. *If the letter comments on a published article in Anaesthesia, please send three copies; otherwise two copies of your letter will suffice.*

other pain is also present seems a poor justification for not bothering to minimise morbidity from the anaesthetic, as the authors suggest.

Our study,<sup>1</sup> quoted by the authors as a failure of benzodiazepine pretreatment, demonstrated a very worthwhile reduction of suxamethonium myalgia by pretreatment with a nondepolarising drug. We had been careful to separate myalgia from obvious abdominal wall and shoulder tip pain caused by the laparoscopy itself, from that caused by suxamethonium. We do not believe postal questionnaire or telephone enquiry to be valid methods of postoperative assessment. Our recently completed study of daycase dental patients involved domiciliary postoperative assessment.<sup>2,3</sup> Only in this way is it possible to separate operative morbidity from that resulting from anaesthesia. Furthermore, much additional information about other aspects of postoperative morbidity in daycase patients can be obtained which would be otherwise unobtainable by other methods.

Royal Preston Hospital,  
Preston PR2 4HT

A. S. LAURENCE  
S. O. SMITH

### References

1. LAURENCE AS. Myalgia and biochemical changes following intermittent suxamethonium administration. Effects of alcuronium, lignocaine midazolam and suxamethonium pretreatments on serum myoglobin, creatinine kinase and myalgia. *Anaesthesia* 1987; **42**: 503-10.
2. SMITH SO, LAURENCE AS, DeSILVA F. Survey of daycase morbidity by domiciliary assessment. *British Journal of Anaesthesia* 1990; **65**: 284.
3. DeSILVA F, SMITH SO, LAURENCE AS. IV dantrolene before daycase suxamethonium use: Domiciliary assessment of morbidity. *British Journal of Anaesthesia* 1990; **65**: 588-9.

### A reply

Telephone inquiry is commonly used to assess postoperative follow-up in ambulatory patients,<sup>1-4</sup> especially in large metropolitan areas. Others<sup>5-7</sup> have used postal questionnaires to assess postoperative myalgias, but we found the response to telephone interview to be greater than relying on patients to mail back study questionnaires. Obviously, if one had the luxury of extra staff and a smaller community than the New York metropolitan area, then domiciliary postoperative assessment might be feasible. We note that the two studies<sup>8,9</sup> cited by Laurence and Smith regarding domiciliary postoperative assessment did not compare it with telephone interview or postal questionnaire.

In our telephone interviews, we were careful to separate out characteristic postlaparoscopic gas pains of the shoulder, neck, and abdominal wall. If, upon persistent questioning, patients continued to complain of shoulder or arm muscle pains, they were given a postoperative myalgia rating of 1 and included in the none/slight myalgia group for data analysis. Patients who required analgesics (commonly, acetaminophen with or without codeine, or ibuprofen) for incisional pain or localised neck and shoulder pain (typical postlaparoscopic gas pains) were excluded from the myalgia rating. In the study reported by Laurence<sup>10</sup> all of the patients received oral lorazepam

premedication, which may by itself attenuate postoperative myalgia.<sup>11</sup>

In response to the issue of comparing the studies of Zahl and Apfelbaum<sup>5</sup> and of Kenefick *et al.*<sup>12</sup> to ours, we would make the following comment. Both studies were conducted in female outpatients undergoing laparoscopy. Zahl and Apfelbaum<sup>5</sup> reported no difference in the incidence of postoperative myalgias between suxamethonium (pretreated with tubocurarine) and vecuronium, where one might expect no myalgias at all, since there were no fasciculations. Kenefick *et al.*<sup>12</sup> compared suxamethonium (pretreated with tubocurarine) and isoflurane with isoflurane alone and also found no difference in the incidence of postoperative myalgia. Our study was conducted in female patients undergoing laparoscopy on an outpatient basis and showed similar results. For these reasons we conclude that female patients undergoing laparoscopy on an ambulatory basis may be a poor model for studying postoperative myalgia in relation to neuromuscular block technique.

Department of Anesthesiology,  
Mount Sinai School of Medicine,  
New York, NY 10029-6574, USA

M.L. MINGUS  
A. HERLICH  
J.B. EISENKRAFT

### References

1. MELNICK P, CHALASANI J, UY NTL, PHITAYAKORN P, MALLET SV, RUDY TE. Decreasing postsuccinylcholine myalgia in outpatients. *Canadian Anaesthetists' Society Journal* 1987; **34**: 238-41.
2. MANCHIKANTI L, GROW JB, COLLIVER JA, CANELLA MG, HADLEY CH. Atracurium pretreatment for succinylcholine-induced fasciculations and postoperative myalgia. *Anesthesia and Analgesia* 1985; **64**: 1010-4.
3. SOSIS M, BROAD T, LARIJANI GE, MARR AT. Comparison of atracurium and d-Tubocurarine for prevention of succinylcholine myalgia. *Anesthesia and Analgesia* 1987; **66**: 657-9.
4. TREPANIER CA, BROUSSEAU C, LACERTE L. Myalgia in outpatient surgery: comparison of atracurium and succinylcholine. *Canadian Journal of Anaesthesia* 1988; **35**: 255-8.
5. ZAHL K, APFELBAUM JL. Muscle pain occurs after outpatient laparoscopy despite the substitution of vecuronium for succinylcholine. *Anesthesiology* 1989; **70**: 408-11.
6. MANCHIKANTI L. Diazepam does not prevent succinylcholine-induced fasciculations and myalgia. *Acta Anaesthesiologica Scandinavica* 1984; **28**: 523-8.
7. BENNETTS FE, KHAHIL KI. Reduction of post-suxamethonium pain by pretreatment with four non-depolarizing agents. *British Journal of Anaesthesia* 1981; **53**: 531-6.
8. DeSILVA F, SMITH SO, LAWRENCE AS. IV Dantrolene before daycase suxamethonium use: domiciliary assessment of morbidity. *British Journal of Anaesthesia* 1990; **65**: 588-9.
9. SMITH SO, LAURENCE AS, DeSILVA, F. Survey of day-case morbidity by domiciliary assessment. *British Journal of Anaesthesia* 1990; **65**: 284.
10. LAURENCE AS. Myalgia and biochemical changes following intermittent suxamethonium administration. Effects of alcuronium, lignocaine, midazolam and suxamethonium pretreatments on serum myoglobin, creatinin kinase and myalgia. *Anaesthesia* 1987; **42**: 503-10.
11. DAVIS AO. Oral diazepam premedication reduces the incidence of post-succinylcholine muscle pains. *Canadian Anaesthetists' Society Journal* 1983; **30**: 603-6.
12. KENEFICK JP, LEADER, A, MALTBY JR, TAYLOR PJ. Laparoscopy: blood gas values and minor sequelae associated with three techniques based on isoflurane. *British Journal of Anaesthesia* 1989; **59**: 189-94.

### Trauma to the posterior pharyngeal wall caused by a laryngeal mask airway

Young children undergoing repeated anaesthesia to facilitate serial cranial radiotherapy are a clinical challenge for the anaesthetist. It has become the practice in this hospital

to use the laryngeal mask airway (LMA) for these cases as this technique may avoid potential morbidity which could occur from daily tracheal intubation. I report a case of

posterior pharyngeal wall trauma resulting from repeated use of the LMA in such a patient.

A 2-year-old girl with Down's syndrome and acute lymphoblastic leukaemia required serial cranial irradiation and adjuvant chemotherapy. The same technique was used for the final 14 general anaesthetics which she received over a period of 25 days. Following intravenous propofol 3 mg/kg, a size 2 laryngeal mask airway was inserted and inflated with 8 to 10 ml of air. Anaesthesia was maintained with the child breathing spontaneously a mixture of isoflurane 1.25% and 60% nitrous oxide with oxygen. The sum duration of time for the 14 anaesthetics was 415 (mean 29) minutes.

After removal of the LMA at the termination of the last anaesthetic, laryngoscopy was performed to remove secretions from the oropharynx. It was noted that a raised band of oedema 5 mm in width traversed the posterior wall of the oropharynx between the two faucial pillars of the palatopharyngeal arch. The epithelium overlying this oedema was white and sloughing. There were no other mucosal lesions visible. The child had been treated previously for oral candidiasis and the mucosal damage may have been augmented by opportunist infection. X rays of the child with the LMA *in situ* demonstrated that this band of mucosal damage corresponded with the area of contact between the posterior surface of the mask and the oropharynx, as shown diagrammatically in Figure 1. The posterior surface of the LMA is relatively rigid and hard. It is possible that this portion of the mask exerts pressure upon the posterior pharyngeal wall with the cuff inflated. The posterior pharyngeal wall would be compressed between the LMA and the adjacent cervical vertebrae. Tracheal tube cuff pressures greater than 40 mmHg have been found to induce mucosal ischaemia.<sup>1</sup>

The LMA may have advantages in children requiring repeated anaesthesia to facilitate radiotherapy as it avoids instrumentation of the larynx and trachea. However, as in this patient, repeated insertion or prolonged use may cause trauma to the posterior pharyngeal wall. Anaesthetists

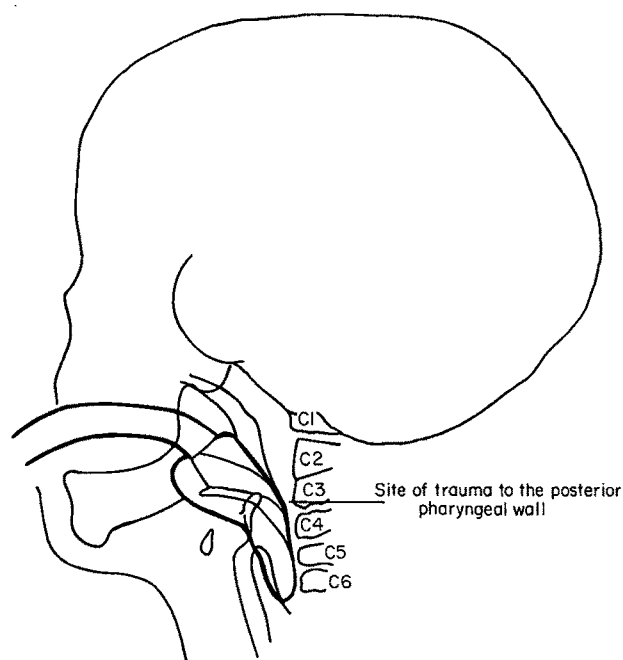


Fig. 1. Diagram of the laryngeal mask airway *in situ*.

should be aware of this possibility and inspection of the oropharynx is recommended during the course of such treatment to determine if damage is developing.

Leicester Royal Infirmary  
Leicester LE1 5WW

R. MARJOT

#### Reference

1. LEIGH JM, MAYNARD JP. Pressure on the tracheal mucosa from cuffed tubes. *British Medical Journal* 1979; 1: 1173-4.

#### Laryngeal mask airway in children: insertion technique

The laryngeal mask airway (LMA) has been used widely in both adult and paediatric anaesthetic practice since its introduction in the mid 1980s. Its insertion is easy, requires little training, and can be used by personnel with varying anaesthetic experience. The most common problem with LMA insertion is difficulty in negotiating the posterior pharynx, despite following the manufacturer's instructions. Some manoeuvres have been suggested to overcome this problem: lateral insertion of the mask, application of firm pressure on the device against the hard palate with the mask tip flattened, pulling the tongue or chin forward or use of a laryngoscope have all been found to be useful. We found that this difficulty can be overcome by inserting the LMA like a Guedel airway. This is achieved by inserting the deflated mask with its lumen facing backwards and then rotating through 180° as it passes downwards into position behind the larynx. This manoeuvre is particularly useful with the size 2 LMA.

We recently conducted a survey on the use of the LMA in 200 children aged 14 months to 14 years. Difficulties with insertion on first attempt were reported in 46 patients, mostly because of inability to insert the mask past the posterior pharyngeal wall, despite correct positioning of the patients' head. In 26 of these patients the problem was overcome by inserting the LMA as if it were a Guedel

airway (24 of these were size 2 masks, and two were size 3) resulting in a clear and unobstructed airway throughout anaesthesia. The rest of the patients needed simple manoeuvres to solve the insertion problem. Six patients had the mask inserted successfully from the side of the mouth, one mask was inserted easily by lifting the jaw. Laryngoscopes were helpful on nine occasions. In two patients, inappropriately sized masks were used initially resulting in failed insertion. In four patients, further attempts using the standard method of insertion enabled the location of the mask correctly.

A pilot study by Dr Brain<sup>1</sup> showed that the patient's airway can be secured safely by inserting the device in a similar fashion in an adult. However, this technique is no longer recommended since it was found later that the epiglottis could become displaced downwards during insertion.<sup>2</sup> Despite this, no respiratory obstruction occurred in the patients and this was thought to be because most of them were female and therefore had smaller epiglottides, which would not cause obstruction even when displaced.

Infants and young children have a larger tongue in relation to the mandible than adults. The epiglottis is also larger and floppier and may lie against the posterior wall of the pharynx. One might expect the technique we suggest to exacerbate these anatomical differences and compromise



the patency of the airway by displacing this larger epiglottis backward, thus obstructing the laryngeal inlet. The final position of the epiglottis could have been confirmed with a fiberoptic laryngoscope, but this was considered unjustifiable since none of the patients had clinical evidence of respiratory obstruction. We confirm that adhering to the manufacturer's instructions facilitates easy insertion of the LMA in most of our patients. However, when difficulty is encountered, inserting the mask in a manner similar to a Guedel airway seems to be an effective alternative manoeuvre, especially in paediatric patients.

Queen Elizabeth Hospital,  
Birmingham B15 2TH

B.F.M. CHOW  
M. LEWIS  
S.E.F. JONES

#### References

1. BRAIN AIJ. The laryngeal mask. A new concept in airway management. *British Journal of Anaesthesia* 1983; 55: 801-4.
2. BRAIN AIJ, MCGHEE TD, MCATEER EJ, THOMAS A, ABU-SAAD MAW, BUSHMAN JA. The laryngeal mask airway. Development and preliminary trials of a new type of airway. *Anaesthesia* 1985; 40: 356-61.

#### Laryngeal masks and chair dental anaesthesia

Chair dental anaesthesia is still practised in many hospitals throughout the country, usually in children. Success depends on the skill and cooperation of the dentist and the anaesthetist. The technique requires the maintenance of both anaesthesia and the airway by a nasal mask, while the dentist performs rapid surgery. A mouth pack is inserted to protect the airway from foreign matter. It also produces an airtight seal, thus aiding in control of depth of anaesthesia. However, this technique may be fraught with difficulties. The ability to maintain a clear airway depends on the patency of the nasal airway, often a problem with children who may have associated upper respiratory tract illnesses. Introduction of nasopharyngeal airways may produce unwanted haemorrhage. The correct size of the mouth pack is difficult to predict and its incorrect placement may lead to airway obstruction.

We would like to recommend a technique using the Brain laryngeal mask airway (LMA) which we have been practising in our hospital for several months. We feel it overcomes many of the difficulties associated with these procedures and provides a practical and perhaps safer method of anaesthesia for such patients. The LMA has been described during dental anaesthesia,<sup>1</sup> but as yet it has not been described for routine use in chair dentistry. The correct placement of the mask should protect the larynx from blood and foreign matter. Use of the mask also results in an unobstructed airway and an airtight seal, allowing depth of anaesthesia to be maintained. The dentist usually has little difficulty in working around the LMA, since

proper positioning at the head in order to facilitate surgery is easier. The head may be safely manipulated and speed of surgery is of much less importance since anaesthesia may be continued indefinitely.

The mask is inserted after intravenous induction. A size 3 LMA is generally most suitable for all children beyond the infant age group, although a size 2 may also be successfully used. Anaesthesia can be maintained with nitrous oxide, oxygen and a volatile agent. We commonly insert a mouth pack as well to help collect any debris. At the end of the procedure, the pack and the mask are removed, secretions are cleared by suction and a Guedel airway is inserted. The child is placed in the lateral position and allowed to recover.

We have found that the technique has been readily accepted by both dentists and anaesthetists. It is a good alternative method of producing ideal conditions for surgery and makes chair dental anaesthesia safer and perhaps less stressful.

Royal United Hospital,  
Bath BA1 3NG

H. NOBLE  
D. J. WOOLLER

#### Reference

1. ALLEN JG, FLOWER EA. The Brain laryngeal mask. An alternative to difficult intubation. *British Dental Journal* 1990; 168: 202-4.

#### Yet another use for the laryngeal mask airway

We have used the paediatric sizes of the Brain laryngeal mask airway (LMA) as an aid to anaesthesia for diagnostic flexible bronchoscopy in children. In our institution, two methods have previously been used. The first method involved using a conventional Rendell-Baker facemask with a swivel connector attached. After the child is anaesthetised and the airway sprayed with lignocaine, the mask is held away from the face and the flexible bronchoscope is inserted through the rubber diaphragm into the nose. The mask is then re-applied to the face while the procedure takes place. The second method is to use a small plain tracheal tube as a nasopharyngeal airway to maintain oxygenation and anaesthesia while the bronchoscopy takes place.

It is generally necessary to give the operator a good view of the structure and function of the laryngeal opening during spontaneous respiration, but it is equally important to maintain a patent airway both to ensure oxygenation and continued anaesthesia. The LMA offers several advantages.

It is usually easy to insert and position, thus freeing the hands of the anaesthetist and giving the operator more freedom of movement. It avoids nasal trauma and the possibility of epistaxis. A relatively good seal around the laryngeal aditus can be obtained and this, together with a good fitting rubber diaphragm in the swivel connector, allows the reservoir bag on the breathing attachment to fill and respiration to be easily visible. Ease of scavenging is another major advantage. The bronchoscopic view through the laryngeal mask is good, allowing dynamic assessment of the airway and vocal cord movement during lightening of anaesthesia. It also permits the use of diagnostic CPAP if required. Therefore, we recommend the use of the LMA for this often difficult procedure in children.

Westmead Hospital,  
Sydney NSW 2145,  
Australia

R.W.M. WALKER  
D. MURRELL

### Unexpected position of the laryngeal mask airway

The laryngeal mask airway (LMA) has been used extensively at our institution for anaesthesia in paediatric radiotherapy. If cranial radiotherapy is required, lateral X rays

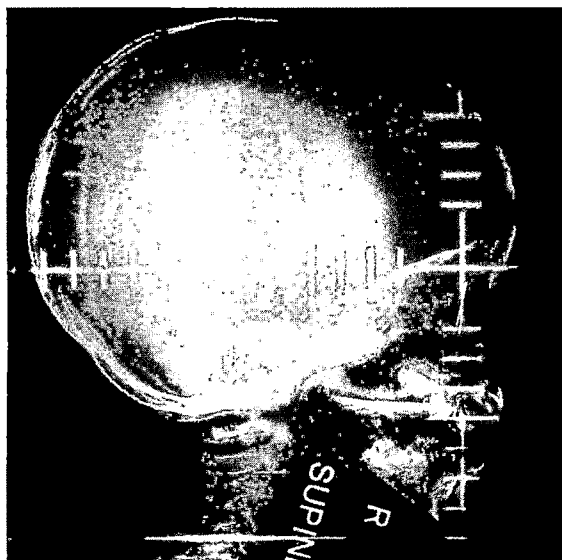


Fig. 1.

are taken at the initial planning stage and these show quite clearly the position of the LMA. I would like to report a case in which the LMA was noted to be in an unexpected position.

A 3-year-old boy with acute lymphoblastic leukaemia was scheduled for planning under general anaesthesia before cranial radiotherapy. Anaesthesia was induced with nitrous oxide, oxygen and halothane via an Ayre's T-piece and a size 2 LMA inserted. The cuff was inflated with 5 ml of air and clinically the presence of a good airway noted. The patient was monitored by pulse oximetry and remote-controlled camera. The planning and initial treatment were completed. The LMA was removed and the patient recovered uneventfully. On review of the planning X rays, it was noted that the LMA had been placed in the mouth with the distal part of the cuff positioned against the posterior wall of the oropharynx (Fig. 1). The planning X rays of 14 other patients were reviewed and the LMA in all cases had been placed in the correct position.

This case illustrates that radiographs can reveal the position of an LMA after insertion and that even in the presence of a good airway, the LMA may not be correctly placed.

St. Bartholomew's Hospital,  
London EC1A 7BE

A.R. MOLLOY

### Aspiration pneumonia and the laryngeal mask airway

We are concerned by the case report from Drs Griffin and Hatcher describing aspiration pneumonia in a young woman following cholecystectomy (*Anaesthesia* 1990; 45: 1039-40). We must presume that at the time they chose to use a laryngeal mask airway they were unaware of the manufacturer's recommendations. The case report illustrates well the dangers of using a new technique on the basis of limited anecdotal reports. Great care is needed if the development of anaesthetic equipment and techniques is to avoid being hampered by expensive and restrictive bureaucratic controls which may be imposed if the current processes are perceived to expose patients to unacceptable hazards.

We wish to draw readers' attention to a study performed some years ago in this hospital,<sup>1</sup> which showed that many routinely prepared patients have stomach contents that place them at risk of acid aspiration and that this is more likely in patients having upper abdominal surgery (approxi-

mately 60% of 23 such patients having volumes in excess of 40 ml and pH < 2.5). In addition, the tendency of patients undergoing biliary tract surgery to secrete large volumes of gastric juice during the procedure was noted. Prior treatment of the patient described in this case report with H<sub>2</sub> blockade and metoclopramide would have reduced the risks of adverse consequences whatever form of airway maintenance was used. The reputation of the laryngeal mask is not well served by inappropriate use.

Lewisham Hospital,  
London SE13 6LH

A.W. PEARCE  
M.L. HEATH

### Reference

1. HESTER JB, HEATH ML. Pulmonary acid aspiration syndrome: should prophylaxis be routine? *British Journal of Anaesthesia* 1977; 49: 595-9.

### Malfunctioning of disposable breathing systems

Recently, we bought some disposable breathing systems manufactured by Intersurgical, Middlesex, UK and designed for use with Bennet/Bourns ventilators. We would like to report a potentially lethal manufacturing defect in a few of these systems.

When we connected one of the breathing systems to the ventilator, we noticed that the expiratory valve was leaking at 40 cmH<sub>2</sub>O and the system pressure never fell below 30 cmH<sub>2</sub>O after the first breath in the test lung. The ventilator functioned normally when connected with the original breathing system provided with it. A quick check of the system did not reveal any obvious occlusion or kink. Since the expiratory valve (Fig. 1) was malfunctioning, we



Fig. 1.

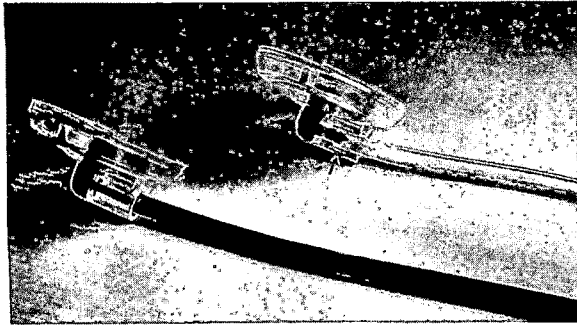


Fig. 2.

dismantled it and found a block in the tube which delivers gas to the pressure chamber over the expiratory valve, which is meant to keep the valve closed during the inspiratory phase. This block in the tubing never allowed the air to be displaced from the pressure chamber into the tubing during expiration and thus kept the valve closed even during expiration. This block has been demonstrated by instilling the dye into the dome of the expiratory valve. The dye did not flow beyond the block in the tubing of the malfunctioning system (Fig. 2), whereas it traversed the whole length of the tubing in the normally functioning system. This is a manufacturing defect which has been overlooked.

This prompted us to check the rest of the disposable breathing systems supplied to us by the same company and we found the same fault with half of them. We wrote to the manufacturer who stated that no similar previous problems had been reported to them and they have subsequently

improved their quality control. We would like to alert our colleagues to the possibility of a malfunctioning expiratory valve in these breathing systems. A thorough check should be made before connecting them to a patient to avoid the potentially catastrophic complication of barotrauma.

Jipmer,  
Pondicherry-605006,  
India

S. SHOREY  
M. RAVISHANKAR

#### A reply

Intersurgical were made aware of this problem by Dr M. Ravishankar in March 1990, when the incident occurred. Two systems were found to be malfunctioning as a result of blockage, which could have been due to the improper adhesion of the flow tube to the manifold body.

Intersurgical confirm that there were no other problems reported of this nature and because the defective samples were never returned to Intersurgical for examination, a full report as required by GMP recommendations, could not be carried out by the company. Dr Ravishankar was advised to return the sample to Intersurgical for examination.

As a result of the written complaint, Intersurgical improved the written work instructions covering the assembly of the manifold valves and instituted 100% inspection and testing. Intersurgical would agree with Professor Shorey and Dr Ravishankar that a thorough check of all systems should be made before a product is used on a patient.

Intersurgical Ltd.  
Twickenham  
Middlesex TW2 6RS

S.K. WILLIAMS

#### Pneumatic tourniquets

Dr P.J. Mills (*Anaesthesia* 1991; 46: 229-30) has performed a valuable service in drawing the attention of anaesthetists to the extremely unsatisfactory design of the majority of pneumatic tourniquets in use in hospitals in the UK. The British Standard Specification for Pneumatic Tourniquet Equipment (BS 7088:1989) was drawn up to encourage the manufacture of safer tourniquet equipment. The committee responsible for its production made a determined effort to produce a standard that would minimise all the known hazards associated with inadequate tourniquet design; particular attention was given to those features which appeared to have contributed to a series of fatal accidents associated with intravenous regional analgesia (IVRA).<sup>1</sup> The problem referred to by Dr Mills is covered by a requirement for pressure gauges to have pointer stop pins; the pins were to be positioned on the ungraduated portion of the dial (so that zero errors are also apparent). In addition there are, amongst others, requirements to ensure even distribution of pressure under the cuff, to avoid excessive pressure or accidental deflation and to eliminate errors in interpretation of markings.

Many anaesthetists believe that the tourniquet is the responsibility of the surgeon when it is not being used as

part of an anaesthetic technique; however, I believe it is worth reflecting that it is the anaesthetist who has deprived the patient of their protective reflexes and should therefore share the responsibility for ensuring that no trauma (other than the surgical procedure which has been consented to) is inflicted on him or her. It would appear that patient safety is insufficient as an incentive to manufacturers; market advantage is also required and this depends on anaesthetists, surgeons and supplies officers obtaining and studying the standard. So far, I am unaware of any equipment claiming compliance. BS 7088 will only achieve its objective if all concerned in the selection and purchase of tourniquet apparatus seek the manufacturers' compliance with it.

BS 7088:1989 is available from British Standards Institution, 2 Park Street, London W1A 2BS.

Lewisham Hospital,  
London SE13 6LH

M.L. HEATH

#### Reference

1. HEATH ML. Deaths after intravenous regional anaesthesia. *British Medical Journal* 1982; 285: 913-14.

#### Cuff failure—a complication of tracheal intubation

In past years it was the practice to test cuffs of re-usable red tracheal rubber tubes before use and to avoid cuff failure, after insertion into the trachea. Present day plastic cuffed tubes, each removed from a sealed packet for single use

only, have perhaps led to relaxation of this previous routine. Recent reviews of the complications associated with tracheal intubation do not mention cuff failure. This may indicate its rarity nowadays, but surely ignores its

significance of exposing the patient to the serious, even dire, consequences of inadequate ventilation and regurgitation-aspiration.

We have had a number of cuff failures resulting in deflation, including deflation during anaesthesia, 2 hours after a maxillary osteotomy, with resultant aspiration of blood, and one patient with severe ARDS where changing a nasotracheal tube was deferred for 10 days because of anticipated difficulty in re-intubation; this latter incident resulted in hypoventilation, hypoxia and aspiration.

These incidents prompted us to ask several manufacturers about prior testing of tube cuffs and the duration of cuff reliability. In reply all emphasised their standards of quality control including the expensive procedures of inspecting and inflation testing (up to 3 hour periods) each tube. It was felt unrealistic to expect or guarantee 100% reliability on 100% occasions because in any manufacturing process it was possible a few items might escape quality safety nets and leave the factory substandard. One manufacturer, from records and customer feedback, suggested 99.999% reliability could be expected when opening a new undamaged tube. Such a failure rate is similar to the incidence suggested for grade 4 intubation. All advised visual inspection of each tube and inflation testing of its cuff prior to intubation. Medicolegally, it seems important to follow the manufacturer's recommendations, which may be printed on the packet; without prior testing it cannot be known that the cuff, inflating tubing, and valve were serviceable before insertion if subsequent failure occurs. Obviously, neither factory nor anaesthetist will detect some faults. This applied to the maxillary osteotomy patient where the leak was in the inflating tubing proximal to the cuff.

Answers to our second question were less direct, e.g. tube reliability duration is greatly dependent on stresses to which it is subjected. This is left to the doctors' clinical judgment and it is hoped the tubes will be reliable as long as the clinical situation demands. Presumably manufacture

is based on some design durability specifications and results of testing to component failure. There are reports of prolonged functioning of peroral (60<sup>1</sup> days) and pernasal (65<sup>2</sup> and 87<sup>3</sup> days) cuffed tracheal tubes. Tubes are being kept *in situ* and exposed to greater stresses and risks of cuff failures as the timing and performing of routine tracheostomy are questioned.<sup>2,3</sup> Tracheal tubes assume the anatomical curvatures of the patient's airway in contrast to a newly inserted tube, and are changed only if a cuff problem affects ventilation.<sup>3</sup> Tracheal intubation is a very frequent 'narrowest latitude highest risk' procedure. Next to correct placement, cuff competency must be paramount. Besides inspection and cuff testing, relevant factors include tube size (avoid undersize), cuff inflation (avoid overinflation), atraumatic insertion, lubrication, coughing/straining, syringe removal from inflating valve after cuff inflation, appropriate care of the inflating line and valve, tube fixation, head position and head and neck movement. Tubes can be damaged by teeth, Magill's forceps and nasal structures. In the latter instance a surgical gloved finger may protect the cuff<sup>4</sup> or one manufacturer suggests inflating then emptying the cuff as it is smoothed (tapered) proximally. Direct application of lignocaine spray to the cuff can compromise PVC cuff integrity.

King Edward VIII Hospital,  
Durban,  
South Africa

R. WILLIAMSON  
A.M. GORVEN

#### References

1. VOGELHUT MM, DOWNS JB. Prolonged endotracheal intubation. *Chest* 1979; 76: 110-1.
2. GILSTON A. Prolonged nasotracheal intubation. *Anaesthesia* 1982; 37: 209-10.
3. VIA-REQUE E, RATTENBORG CC. Prolonged oro- or nasotracheal intubation. *Critical Care Medicine* 1981; 9: 637-9.
4. WRIGHT PJ. Nasotracheal intubation: another approach. *Anaesthesia* 1986; 41: 1057-8.

#### Precordial stethoscopes

The importance of continuous monitoring of heart and respiration under general anaesthesia cannot be overstressed. Two simple devices, the precordial stethoscope and the oesophageal stethoscope are in common use with monaural or binaural ear pieces. Conventionally, the chest piece of the ordinary stethoscope is strapped to the chest wall with adhesive plaster. However, it tends to become bulky, may get dislodged while positioning the patient and reliable fixation to the chest wall is often difficult. In infants it occupies a large surface area and if the surgery is to be performed in prone position, the large chest piece presses against the chest wall. An external stethoscope with metal parts obstructs radiological pictures of the heart and chest, and watching the heart action on fluoroscopy screen may be confusing.

Oesophageal stethoscopes cannot be used when the surgery to be performed is on the oesophagus. They are invasive, costly, intended for single use, may not be available under certain circumstances and are usually inserted into the oesophagus after induction of anaesthesia, thus depriving the anaesthetist of continuous monitoring of heart and respiratory sounds in such a critical period. They may kink in the pharynx by manipulation, change of patient position or in certain neck positions, thus making the sounds inaudible. Presence of an oesophageal temperature probe and nasogastric tube may also interfere with the monitoring of the heart and respiratory sounds. If the anaesthetic is to be administered via a facemask, then a leak

proof fit may not be obtained without interfering with sound conduction.

We have used ordinary latex, polyvinyl chloride or rubber tubing, easily available in operating theatres, as a precordial stethoscope by closing the distal end with adhesive plaster and making two, 5 mm wide holes, 1 and 3 cm from the distal closed end. This tube has an external diameter of 8.5 mm and an internal diameter of 6 mm. The tube is applied with the holes facing the chest wall and strapped firmly in place with adhesive plaster. The proximal end is connected to the conventional stethoscope tubing and ear piece.

Used in this way, this simple tubing has been found to be very useful in clinical use as a precordial stethoscope. This simple tube stethoscope is soft, lightweight, economical, noninvasive, can be fixed firmly and reliably to the chest wall, occupies only a small area, allows monitoring at a distance from the patient, can be used repeatedly and is especially suitable for the infants. It does not interfere with radiological procedures and can be valuable in remote areas, when dealing with mass casualties and in situations where adequate monitoring facilities may not be available.

Department of Anaesthesiology,  
Postgraduate Institute of  
Medical Education and Research,  
Chandigarh-160012  
India

A. KUMAR  
V.K. GROVER  
I. BHUKAL  
S. SHARMA

## Guide wire embolism

A 77-year-old woman was scheduled for an urgent laparotomy for a suspected perforated duodenal ulcer. Before induction of anaesthesia a triple lumen central venous catheter (Multicath in Flexane Vygon Ltd) was inserted into the right internal jugular vein. The vein was punctured with the supplied cannula and the guide wire passed into the vein. The cannula was removed and the vein dilator threaded over the guide wire; the wire and dilator were advanced down the vein as one. The dilator was then removed and the triple lumen catheter threaded over the guide wire. The latter was removed easily from the catheter and appeared to be complete, with a J still present at the tip. The operation proceeded uneventfully and the patient was transferred to the intensive therapy unit for elective ventilation of her lungs. A routine chest X ray taken in the ITU revealed that a portion of the guide wire remained within the chest (Fig. 1). The presence of this wire had been unsuspected since the removed wire had appeared complete on removal. After a period of stabilisation on the ITU the patient was transferred to the X ray department for removal of the wire, which was achieved via the right femoral vein using a snare technique described by Thomas *et al.*<sup>1</sup>

The wire was returned to the company via the Department of Health Procurements Office and was found to have two faults. There was a failure of the join between the outer coil and the inner safety wire at the J tip, in conjunction with a fracture in the outer coil which had enabled the outer coil to be stripped off the inner safety wire leaving the complete inner wire with its J tip and the outer coil in the patient. Metallurgical analysis concluded that these two separate fractures were due to torsion stress or overload, even though there had been no difficulty in passing or removing the guide wire.

Guide wires are constructed with an outer wire helix terminating in either a welded or soldered polished round tip. These coils may fracture during manufacture or manipulation so most now have an inner safety wire running the length of the outer coil and welded at both ends of the outer coil.<sup>2</sup> Complete separation and embolisation of a segment of guide wire has been described previously,<sup>3</sup> and usually follows the withdrawal of the guide wire through an introducing needle, thereby shearing off the wire on the needle bevel. This guide wire contained an unusual double fault not previously described.

This case further illustrates the necessity of performing a chest X ray after insertion of central venous cannulae and close examination of a guide wire for breaks in the outer

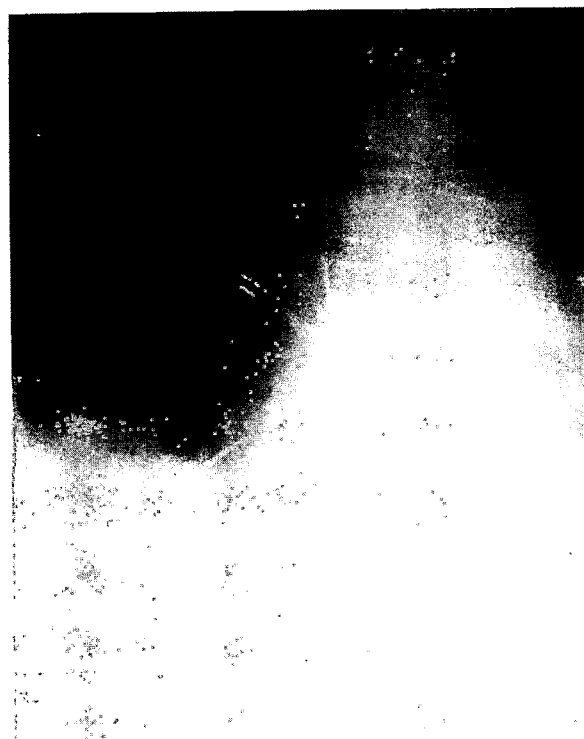


Fig. 1. Chest X ray showing the position of the embolised fragment of guide wire in the superior vena cava, hepatic vein and a loop in the right atrium and ventricle.

coil. Gentle traction on each end of the wire may expose any pre-existing fractures in the outer coil.

Leicester Royal Infirmary,  
Leicester

P. BARKER

## References

1. THOMAS J, SINCLAIR-SMITH BC, BLOOMFIELD D, DAVACHI A. Non-surgical removal of a broken segment of a steel spring guide wire from the right atrium and inferior vena cava. *Circulation* 1964; **30**: 106-8.
2. DOTTER CT, JUDKINS MP, FRISCHE LH. Safety guidespring for percutaneous cardiovascular catheterisation. *American Journal of Roentgenology* 1966; **98**: 957-9.
3. COPE C. Intravascular breakage of Seldinger string guide wires. *Journal of the American Medical Association* 1962; **180**: 1061-3.

## Upper airway obstruction by enlarged tonsils

We read with interest the case report by J.R. Livesey *et al.* concerning a child with airway obstruction as a result of adenotonsillar enlargement (*Anaesthesia* 1991; **46**: 36-7). Recently, we experienced a similar problem with a child, although our patient behaved differently with regard to induction of anaesthesia and the early postoperative course. The child was a hyperactive 9-year-old boy weighing 30 kg. He had a history of disturbed breathing patterns during sleep. These included stridor, apnoea, snoring and frequent night wakening. He was admitted to hospital with severe tonsillitis. A diagnosis of glandular fever was made and later confirmed. Shortly after admission he developed marked stridor accompanied by drowsiness. If he was allowed to fall asleep, he became apnoeic and had to be

woken rapidly. A pulse oximeter registered an  $SaO_2$  of 90% when awake and 80% when asleep whilst breathing air. He refused supplementary oxygen. His tonsils were grossly enlarged and occupied all the visible oropharynx.

The decision was taken to perform an urgent tonsillectomy. We were unable to induce anaesthesia with halothane in 100% oxygen since the child rapidly became apnoeic and hypoxic despite our use of a variety of oral airways. Eventually we decided to administer thiopentone 150 mg and suxamethonium 50 mg, with the surgeon prepared to proceed immediately to tracheostomy if necessary. Intubation proved to be relatively easy and the tonsils were removed. The adenoids, although enlarged, were not touched for fear of uncontrollable haemorrhage.

The postoperative course was stormy. The stridor persisted even in the absence of tonsils. Again, the  $\text{SaO}_2$  varied from 80% when asleep to 90% when awake, despite supplementary oxygen and an oropharyngeal airway. This state of affairs lasted for 12 hours overnight, but by morning the child was no longer stridorous whilst awake. The  $\text{SaO}_2$  rose to 99%. However, it took two further days for the sleep-stridor-apnoea pattern to resolve completely. A month later the parents reported that the child had lost his disturbed sleep patterns.

We were puzzled and disappointed by the persistence of obstructed breathing after tonsillectomy. Possibly it was the result of palatopharyngeal dysfunction associated with

long-standing adenotonsillar hypertrophy and aggravated by the fact that the adenoids were still *in situ*. Whatever the cause it should be borne in mind that tonsillectomy does not always immediately relieve upper airways obstruction caused by enlarged tonsils. The other lesson we learnt from our patient is that inhalation induction in young patients with a history of sleep apnoea is sometimes impossible and preparations should be made in advance to secure the airway by alternative means if necessary.

Colchester General Hospital,  
Colchester CO4 5JL

W. KONARZEWSKI  
P. WALKER  
A. DONOVAN

### Is the pre-operative removal of dentures necessary?

In our group of hospitals it is the policy to remove patients' dentures during their preparation for surgery. We suspect that this may cause considerable distress, especially to women, who may never have been seen (even by close members of their family) without their dentures *in situ*. We believe that this practice is common in hospitals in the UK, unlike Australasia where, we understand, many hospitals allow their patients to wear their dentures to the operating theatre. In these hospitals patients whose tracheas are to be intubated either remove their dentures themselves before induction, or the dentures are removed by the anaesthetist immediately after induction and before intubation. The dentures are then placed in a labelled container and reinserted by the patient in the recovery area. Patients receiving anaesthesia by mask are allowed to leave their dentures *in situ* for the duration of the anaesthetic.

The reason for removing dentures is said to be that the

anaesthetised patient could swallow them or that they could become dislodged and cause respiratory obstruction. Although we accept that this is possible with partial plates, it is unlikely with a full set of dentures. Many denture wearers sleep happily and safely with their dentures in place, removing them only for cleaning.

In our opinion, permitting patients to wear their dentures to the operating theatre allows them greater dignity and lessens pre-operative distress. Additionally, it overcomes the problem of leakage of gases between the mask and the sunken cheeks of the edentulous patient. We would be interested in other readers' views on the routine removal of dentures pre-operatively.

University Hospital of Wales  
Cardiff CF4 4XW

M. COBLEY  
J. DUNNE

### Coagulation screening before epidural analgesia in pre-eclampsia

We were most interested in the results of the audit of coagulation screens on pre-eclamptic patients conducted by Drs Barker and Callander (*Anaesthesia* 1991; 46: 64-7).

We have recently conducted a similar audit at the Leicester Royal Infirmary Maternity Hospital. This covered a 6-month period between 1 May 1990 and 31 August 1990, during which period 3350 deliveries occurred. During this period 176 coagulation screens were performed. Our findings were similar to those reported by Barker and Callander. No pre-eclamptic patient with a platelet count greater than  $150 \times 10^9/\text{litre}$  was found to have abnormal coagulation. Nine patients had a platelet count between 150 and  $100 \times 10^9/\text{litre}$  associated with severe pre-eclampsia and normal coagulation. Two of these patients had fibrin degradation product (FDP) levels of  $500 \mu\text{g}/\text{litre}$  (normal  $< 500 \mu\text{g}/\text{litre}$ ), a small increase not necessarily associated with the development of a coagulopathy. One patient had a platelet count of less than  $100 \times 10^9/\text{litre}$  associated with coagulation abnormalities and severe pre-eclampsia (prothrombin time 22.1/13.8, PTTK 51/40, fibrinogen level

$0.7 \text{ g}/\text{litre}$ , FDP  $8000 \mu\text{g}/\text{litre}$ ). One patient in the Cardiff audit had a platelet count of  $153 \times 10^9/\text{litre}$  and a prolonged KCCT, the significance of which is uncertain. The remaining patients in this report who had coagulation abnormalities all had a platelet count of less than  $150 \times 10^9/\text{litre}$ .

In conclusion, we would agree that a platelet count is a useful first line test for screening patients with pre-eclampsia, since there is a minimal risk of significant haemostatic abnormality co-existing with a normal platelet count in this group of patients. Reducing the number of unnecessary coagulation screens would prevent delay for patients who require an epidural and save resources; the cost of a coagulation screen in this hospital is approximately £20.

Leicester Royal Infirmary,  
Leicester LE1 5WW

T.N. TROTTER  
J.K. WOOD  
A.L. ARMSTRONG  
A.E. MAY

### Ethyl chloride and diethyl ether for assessing the extent of epidural blockade

I recently wrote to *Anaesthesia News* about the forgotten potential risks of using ethyl chloride for testing the adequacy of epidural blockade. Considerable numbers of

the 50 ml tubes are used throughout the country; this hospital issued 214 in the 1989/90 year, not all to the maternity unit. Two relevant incidents have since then been



SUCH A SENSITIVE AREA ...

"Headaches caused  
by leakage of  
spinal fluid may  
be minimised by  
using... a needle  
without a cutting  
bevel."<sup>1</sup>



NOW AVAILABLE  
IN 25G AND 27G

B-D® FINE GAUGE PENCIL POINT NEEDLES



... DESERVES A TRULY SENSITIVE APPROACH

**BECTON  
DICKINSON**

**Becton Dickinson UK Limited**

Between Towns Road, Cowley, Oxford, OX4 3LY, England.  
Tel: 0865 748844 Fax: 0865 717313

*Reference*

1. Moore DC. Regional Block. A handbook for use in clinical practice of medicine and surgery. 4th ed. Springfield Ill: Charles C Thomas; 1964; 358-359.



## The Sprotte Spinal Needle

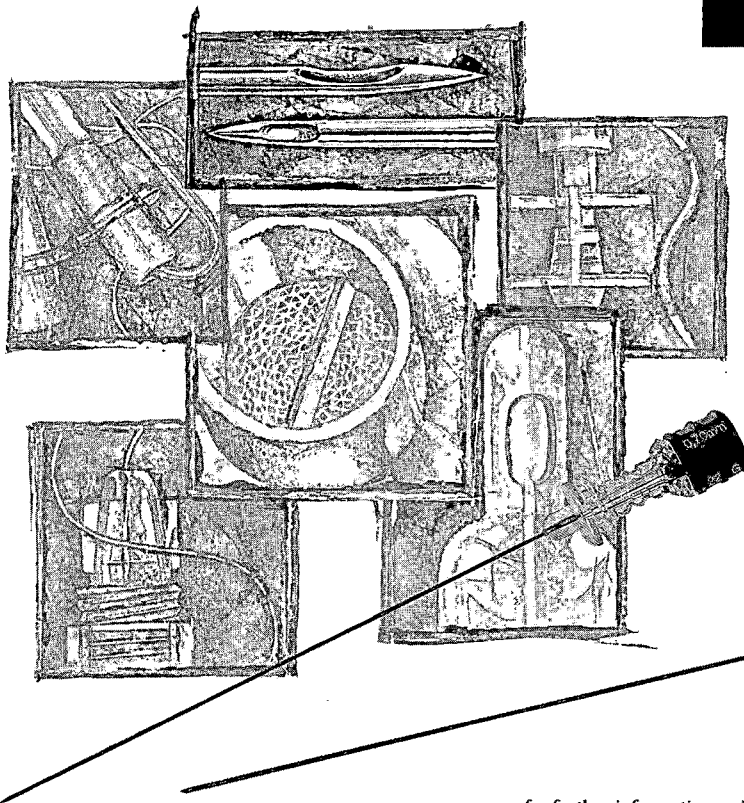
### The new atraumatic spinal needle

- Atraumatic tip displaces dural fibres.
- Dural tap closes on withdrawal of needle.
- Minimises loss of CSF and subsequent post dural puncture headaches.
- Easy injection and aspiration due to the relatively wide lateral eye.

21g, 22g, 24g

Sprotte spinal needles

now available from:



for further information write to:

RUSCH UK Ltd. Freepost, High Wycombe, Bucks. HP12 3BR or Freephone 0800 581121

**Rusch** U.K. Ltd.

THERE MUST BE  
AN EASIER WAY  
TO AUDIT!



### The Synchro Soft Audit System is:-

- Devoid of paperwork
- Quick and simple to use
- Proven, reliable and effective
- Available now
- Used by many major hospitals
- The professional solution

For information or a demonstration, contact

Denise Chamberlain  
Product Information Manager  
Synchro Soft  
Red Lion Cottage  
Catforth  
Preston PR4 0HE

**Tel: (0772) 690546**

**Synchro  
Soft**

reported. In the first,<sup>1</sup> diethyl ether, kept on an anaesthetic machine to test thermal block, was injected epidurally. In the second,<sup>2</sup> ethyl chloride, used to produce local analgesia, actually rendered a patient unconscious!

Both agents are potentially dangerous as sources of a thermal challenge and these incidents underline my proposal that they should be replaced rapidly in obstetric units and operating theatres by picnic box cold blocks, which are safe, cheap and nonpolluting.

St Helier Hospital,  
Carshalton,  
Surrey SM5 1AA

W.A. LINDSAY

#### References

1. MAPPEs A, SCHAEr HM. Accidental injection of ether into the epidural space. *Anaesthesia* 1991; 46: 124-5.
2. SMITH GD, KHALIFA M. Nothing new under the sun. *Journal of the Medical Defence Union* 1991; 7: 11.

#### Assessment of training in anaesthesia and related skills

The introduction of log books into anaesthesia represents one aspect of assessment of training of the junior anaesthetist. However, many log books give no clear idea of skill or of progress and merely record 'exposure' to a skill or technique. Furthermore, training of a single doctor in a single technique, for example tracheal intubation, may be assigned to a number of different anaesthetists; assessment of skill or progress can be difficult when departments are large or geographically spread. The assumption, for

other skills, such as arterial cannulation. 'Adequacy' of training could be defined; for example 20 successful, consecutive intubations might be considered appropriate for 'solo' anaesthesia. However, standards could be set (and altered) at will. 'Proof' of practical skills is now required before entry to at least one national professional examination, viz. skill in basic cardiac life support for MRCGP, or it may be required locally, for example for nurse training in intravenous cannulation. The method demonstrated gives

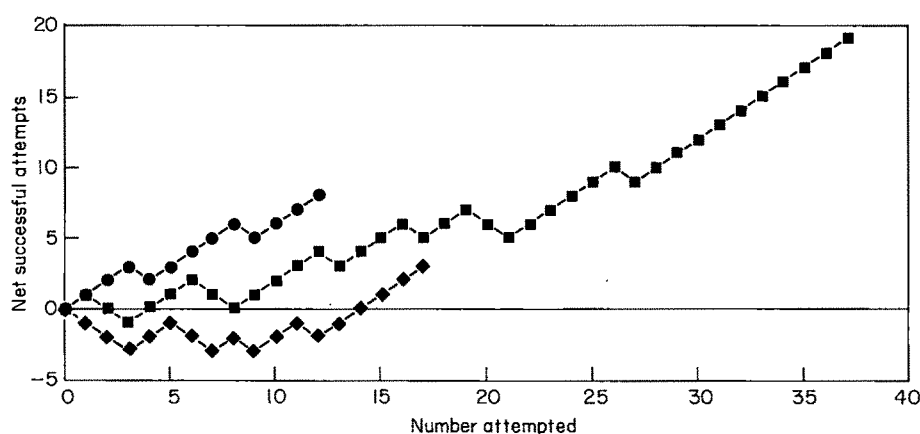


Fig. 1. Graphical demonstration of adequacy of training and skill acquisition. The graph shows excellent skill in central cannulation, possibly the result of previous training. However, the doctor requires significant help with arterial cannulation, shown by repeated failures. ■, tracheal intubation; ♦, arterial cannulation; ●, central cannulation.

instance, that 2 months' exposure to routine anaesthetic practice is adequate, is simplistic.

We have recently utilised a graphical technique to assess progress (Fig. 1). Successful completion of a manoeuvre (or failure) is indicated on the ordinate, while number of attempts is indicated on the abscissa. A 'success' moves the point diagonally upwards, whilst a 'failure' moves the point diagonally downwards. Reasons for failure could be indicated. Although we have used this only for intubation skills, the method can, as shown, be used equally well for

an unambiguous record of training, both in quantity and quality.

We claim no originality for the method of assessment demonstrated above, but we believe it deserves wider publicity.

South Cleveland Hospital,  
Middlesbrough TS4 3BW

P.G. LAWLER  
V.R. PATLA  
E. GARCIA  
N. PUTTICK

#### Block of epidural needle by blood clots

Pregnancy is known to be associated with a hypercoagulable state and recently we encountered two patients in whom blood clots formed in a Touhy needle. Both had requested epidural analgesia for relief of labour pains.

The first patient had a Touhy needle inserted up to the 3 cm mark. The stylet was removed and the needle was advanced employing the 'loss of resistance' to air technique. At the 4 cm mark, blood was noted at the hub of the needle. A decision was made to proceed 1 minute later, but at the 5 cm mark there was still no 'loss of resistance' to air. Since most of our local patients should have the extradural space identified within 5 cm, it was decided to withdraw the

needle. Inspection showed that the tip was completely occluded by a blood clot. The procedure was repeated and the patient subsequently had an uncomplicated epidural analgesia established via a different lumbar interspace. In the second patient the extradural space was located at the 4.5 cm mark, but the catheter filled with blood on insertion. This was removed and the procedure repeated via the same lumbar interspace. However, when the needle was advanced to the 5 cm marking, there was still no 'loss of resistance' to injection of air. The needle was withdrawn and the tip was found to be completely occluded with a blood clot. She subsequently had successful epidural analgesia, but after

delivery developed a headache consistent with a dural puncture.

We have since advised our colleagues to exercise caution and a high index of suspicion whenever blood is noted whilst trying to locate the extradural space with the Touhy needle, especially in the obstetric patients. Perhaps the use

of saline rather than air might have prevented clot formation.

Department of Anaesthesia,  
National University of Singapore,  
Singapore 0511

E.T.M. LIM  
T.S. TEH  
Y.H. LI

#### Ketamine, midazolam and vecuronium infusion

We read with great interest the case report of Drs Riley and McBride (*Anaesthesia* 1991; 46: 122-3) on the use of total intravenous anaesthesia with a ketamine, midazolam and vecuronium infusion for a patient with Down's syndrome and Eisenmenger's syndrome. They considered that slow emergence was an advantage but possibly excessively long.

This anaesthetic technique,<sup>1</sup> modified by a rapid sequence induction using suxamethonium bromide, was correctly used under field conditions in the Gulf. The problem of prolonged recovery was discussed at a Desert anaesthesia symposium in January attended by anaesthetists from the British Army, US Navy and US Marine Corps. After considering the pharmacokinetics of this combination, it was thought that the original infusion rate should be reduced by one third after one hour and, as before, the infusion stopped 10 minutes before the end of the procedure.

During the first month of the Gulf conflict, two patients underwent extensive debridement following trauma.

Anaesthesia lasted 111 and 113 minutes, which is a similar duration to Drs Riley and McBride's case. Total intravenous anaesthesia as described above was used, the infusion rate was reduced by one third after one hour and stopped about 10 minutes before the end of surgery. Extubation was possible 2 and 3 minutes after reversal and the patients were awake and able to answer questions, 22 and 29 minutes after the infusion was stopped.

We would welcome further reports of prolonged use of this technique under field conditions.

Cambridge Military Hospital,  
Aldershot,  
Hants GU12 2AN

P.J. WARD  
J. RESTALL

#### Reference

1. RESTALL J, TULLY AM, WARD PJ, KIDD AG. Total intravenous anaesthesia for military surgery. A technique using ketamine, midazolam and vecuronium. *Anaesthesia* 1988; 43: 46-9.

#### Blood clot obstruction of the upper airway

A 32-year-old female patient with previously diagnosed acute lymphoblastic leukaemia presented with a 12-hour history of intermittent difficulty in breathing, precipitated by lying flat. On examination, no significant abnormalities were found when she was sitting, but spasmodic and severe inspiratory stridor occurred when she was lying horizontal. A plain lateral neck radiograph revealed a mass in the supraglottic region (Fig. 1). Indirect laryngoscopy revealed a mobile pale fibrinous mass arising from the posterior pharyngeal wall, overlying the glottis and moving forward to partially obstruct it on inspiration. She was scheduled for

direct laryngoscopy and as her platelet count was less than 35 000, 10 units were transfused pre-operatively.

Following pre-oxygenation, anaesthesia was induced with halothane in oxygen with the patient in the sitting position. Anaesthesia was deepened, and the patient gradually laid flat; no respiratory obstruction occurred, and laryngoscopy revealed a firm, mobile mass with a pedunculated base, arising from the posterior pharyngeal wall. The trachea was intubated easily with a 6.0 mm cuffed micro-laryngoscopy tube. The mass was removed with minimal haemorrhage and the patient made a rapid and uneventful recovery. Histology revealed that it was a fibrinous blood clot.

The occurrence of ball-valve obstruction in the bronchus leading to inspiration with difficulty, but an inability to exhale was described,<sup>1</sup> and tumours are well recognised as a cause of obstruction.<sup>2</sup> Obstruction from aspergillomata in the airways of immunocompromised patients was described recently.<sup>3</sup> The obstruction in our patient was supraglottic and was the result of a mobile, but firmly adherent clot in a patient with thrombocytopenia secondary to leukaemia. Whilst this is a very rare cause of obstruction, it should be borne in mind in patients with clotting abnormalities.

St. Mary's Hospital,  
London W2

I.A. EWART  
M. WESTON

#### References

1. KRUCZEK ME, HOFF B, KESZLER BR, SMITH B. Blood clot resulting in ball-valve obstruction in the airway. *Critical Care Medicine* 1982; 10: 122-3.
2. TODRES DD, REPPERT SM, WALKER PF, *et al.* Management of critical airway obstruction in a child with a mediastinal tumour. *Anesthesiology* 1976; 45: 100.
3. BERLINGER NT, FREEMAN TJ. Acute airway obstruction due to necrotizing tracheobronchial aspergillosis in immunocompromised patients: a new clinical entity. *Annals of Otolaryngology and Laryngology* 1989; 98: 718-20.



Fig. 1.

SUCH A SENSITIVE AREA ...

"Headaches caused  
by leakage of  
spinal fluid may  
be minimised by  
using a smaller  
gauge spinal  
needle ..."<sup>1</sup>

NOW AVAILABLE  
IN 27 AND 29  
GAUGE

B-D® FINE GAUGE SPINAL NEEDLES



... DESERVES PRECISION AT YOUR FINGER TIPS

**BECTON  
DICKINSON**

**Becton Dickinson UK Limited**

Between Towns Road, Cowley, Oxford, OX4 3LY, England.  
Tel: 0865 748844 Fax: 0865 717313

*Reference*

1. Moore DC. Regional Block. A handbook for use in clinical practice of medicine and surgery. 4th ed. Springfield III: Charles C Thomas; 1964; 358-359.



# Canadian Journal of Anaesthesia

**Editor-in-Chief**  
David R. Bevan  
*Montreal, Quebec*

**Editor Emeritus**  
R.A. Gordon  
*Toronto, Ontario*

**Editorial Board**  
Alan D. Baxter  
*Ottawa, Ontario*

A.O. Davies  
*North Bay, Ontario*

François Donati  
*Montreal, Quebec*

Peter G. Duncan  
*Saskatoon, Saskatchewan*

Geoffrey L. Dunn  
*Hamilton, Ontario*

Jean-François Hardy  
*Montreal, Quebec*

R.J. Hudson  
*Winnipeg, Manitoba*

Richard L. Knill  
*London, Ontario*

R. Kozody  
*Edmonton, Alberta*

Henry I. Manson  
*St. John's, Newfoundland*

René Martin  
*Sherbrooke, Quebec*

Brian Milne  
*Kingston, Ontario*

William H. Noble  
*Toronto, Ontario*

Sheldon H. Roth  
*Calgary, Alberta*

David J. Stewart  
*Vancouver, British Columbia*

Claude Trépanier  
*Quebec City, Quebec*

The Canadian Journal of Anaesthesia, published by the Canadian Anaesthetists' Society, is a highly regarded publication now in its 38th year. Formerly the Canadian Anaesthetists' Society Journal (Volumes 1-33, 1954-1986) the journal acquired both a new name and format in 1987.

Regular features include reports of investigations, informative review articles, clinical and technical reports, editorials, book reviews, correspondence, classified ads and meeting notices.

The CJA is published 8 times per year (January, March, April, May, July, September, October and November) plus the Annual Meeting Supplement which accompanies the May issue. This supplement contains refresher course outlines and abstracts of papers presented at the Society's Annual Meeting.

International in content and readership, consider the Canadian Journal of Anaesthesia for authoritative, up to date information on anaesthesia and related medicine.

---

## 1991 Subscription Information

Volume 38 (8 issues plus Annual Meeting Supplement) 1991  
Individuals: CAN & US \$115.00 / FOREIGN \$135.00 + AIR MAIL (optional): \$65.00  
Institutions: CAN & US \$150.00 / FOREIGN \$170.00 + AIR MAIL (optional): \$65.00

All subscriptions begin with the first issue of the calendar year.

Cheques should be made payable to the Canadian Journal of Anaesthesia and must be drawn on a Canadian or US bank account. The prices quoted are in Canadian dollars. Visa, Mastercard and American Express are accepted, please include expiry date.

ISSN: 0832-610X

Sample copies are available.

## Abstracted or indexed in:

Excerpta Medica, TSD Index Medicus; and available on microfilm through University Microfilms.

---

Please send cheques or information requests to:

**Canadian Journal of Anaesthesia**  
187 Gerrard Street East  
Toronto, Ontario, Canada M5A 2E5

Telephone: (416) 923-1449 Fax: (416) 944-1228

### Capnography and sticking valves in the circle system

The report by Parry, Jewkes and Smith (*Anaesthesia* 1991; 46: 229) highlights the importance of capnography during anaesthesia using the circle system and potential design problems with absorber valves and associated valve-cages. The quest for low mass in the expiratory valve structure renders them liable to sticking to the inevitable water droplets found condensed in the expiratory valve cage. Whalley<sup>1</sup> reports sticking of the mica expiratory valve in an Ohmeda series 5 absorber, which was then redesigned to eliminate this problem. Two new BOC absorber units incorporating lightweight valves purchased by the Royal Melbourne Hospital; both allowed rebreathing due to expiratory valve incompetence when water condensed on the valve cage. This was rectified by changing the valve discs to the heavier metal discs which were also available at the time from CIG, Australia. An Ulco absorber unit incorporating a silicon Ambu valve has also been reported to allow rebreathing,<sup>2</sup> as has a CIG twin cannister unit due to faulty maintenance.<sup>3</sup> Unfortunately, the authors did not specify the absorber involved in their incident. However, it would appear that many units available have allowed rebreathing on occasions.

Two design areas appear to be involved in these events. In the case of disc valves, the metal cage surrounding the valve needs to have minimal contact with the disc. This is usually in the form of small pointed projections on the cage undersurface to reduce droplet formation. In the case of

'fish mouth' or 'flutter valves', the dome should be designed to discourage droplet formation by utilising a concave surface allowing sufficient clearance from the valve cusp travel. The capnographic appearance of incompetent expiratory valves is well documented in the literature.<sup>2,4</sup> I think the authors' closing comment could perhaps be extended to cover the benefits of capnography during all forms of general anaesthesia, since no breathing system is immune to mechanical problems.

Nuffield Department of Anaesthetics, F.A. ROSEWARNE  
The Radcliffe Infirmary,  
Oxford OX2 6HE

### References

1. WHALLEY DG. Malfunctioning unidirectional valves of Ohmeda series 5 & 5A carbon dioxide absorbers. *Canadian Journal of Anaesthesia* 1988; 35: 668-9.
2. ROSEWARNE FA, NUNN BJ. Expiratory valve failure. *Anaesthesia and Intensive Care* 1990; 18: 273-4.
3. ROSEWARNE F, WELLS DG. Three cases of valve incompetence in a circle system. *Anaesthesia and Intensive Care* 1988; 16: 376-7.
4. BERMAN LS, PYLES ST. Capnographic detection of anaesthesia circle valve malfunctions. *Canadian Journal of Anaesthesia* 1988; 35: 473-5.

### 32-gauge spinal catheters through 26-gauge needles

I read with interest the report by Drs Nagle, McQuay and Glynn on their experiences with the use of a 32-gauge Micro-Spinal Catheter through 26-gauge needles (*Anaesthesia* 1990; 45: 1052-54). They state that kinking of the catheter regularly occurs and that resistance/difficulty threading the catheter was a 'repeated and repeatable phenomenon', with no details of the actual number of times this occurred or the number of patients in whom the spinal catheter passed uneventfully.

In my own very small series of two patients in whom I have attempted to use the 32-gauge catheter, there was a 100% failure to thread it beyond the end of the spinal needle into the subarachnoid space. This is in marked contrast with my experience of the 28-gauge spinal catheter ('Cospan' Kendal), which I have used in 11 patients and have experienced no difficulties with inserting it. I would be most grateful if Drs Nagle *et al.* could state in how many of the 28 patients studied was it impossible to thread a 32-gauge catheter and in what percentage of patients were difficulties encountered.

Queen Elizabeth Hospital,  
King's Lynn PE30 4ET

N.M. DENNY

### A reply

Thank you for the opportunity of answering Dr Denny's letter. We were able to place the 32-gauge spinal catheters

easily in about one third of patients, with some difficulty in another third and we failed to place the catheter in about a third of our attempts. Recently, Drs Kestin and Goodman (*Anaesthesia* 1991; 46: 93-4) reported their experience in using these catheters in 13 elderly males. They failed to achieve satisfactory anaesthesia in four patients, either because of kinking of the catheter or because of difficulty in placement. The same group also describe using the catheters for spinal anaesthesia for Caesarean section,<sup>1</sup> where they failed in only two out of 20 patients. Their mean time to place the catheter was 3 minutes, but the range of 1-45 minutes suggests that they too experienced difficulties in some patients. They used an oblique paraspinal approach in all their patients and this may allow the catheter to thread into the subarachnoid space more easily than the midline approach which we used. They make the comment that 'success improves considerably with experience'; we would certainly endorse this.

Abingdon Hospital,  
Abingdon,  
Oxon OX14 1AG

C.J. NAGLE  
H.J. MCQUAY  
C.J. GLYNN

### Reference

1. KESTIN IG, MADDEN AP, MULVEIN JT, GOODMAN NW. Comparison of incremental spinal anaesthesia using a 32-gauge catheter with extradural anaesthesia for elective Caesarean section. *British Journal of Anaesthesia* 1991; 66: 232-6.

### Suxamethonium in a cannula deadspace—a danger

We have observed three brief episodes of asystole and one episode of profound sinus bradycardia in children. It is likely that a very small dose of suxamethonium was responsible.

Three children undergoing ENT or dental surgery were fit, aged 14-15 years and weighed 40-53 kg. The fourth child, who experienced the profound bradycardia, was aged 3 years, weighed 15 kg and was anaesthetised for urgent



manipulation of a fractured forearm. Venous access was established using a 25-G Butterfly needle (Abbott) in two children, and Venflon cannulae (20 and 25 G) in the other children. Anaesthesia was induced with thiopentone (4–5 mg/kg) followed by suxamethonium (1.3–2.0 mg/kg) and maintained with 1–2% isoflurane in nitrous oxide and at least 33% oxygen. All children were monitored with an electrocardiogram and a pulse oximeter. Some 5–10 minutes after induction, with pulse rates of 90–143 beats/minute, saline, pethidine or dexamethasone was injected through the needles or cannulae. A few seconds later the pulse oximeter alarm sounded and the ECG showed bradycardia. This progressed to asystole in the three older children which lasted about 10 seconds before normal rhythm was restored spontaneously. In the fourth child sinus bradycardia of 40 beats/minute was treated with atropine 180 µg before it could progress.

It is well known that a second dose of suxamethonium will produce a bradycardia or even asystole if given to a patient who has not received an anticholinergic drug.<sup>1</sup> It seems most likely that in our cases it was the flushing of suxamethonium contained within the cannula or needle deadspace that was the cause of the bradycardia or asystole. The critical size of the second dose of suxamethonium and the critical dose interval has not been defined. Usually the effect is seen with a second dose as large as the first, given as respiration returns in order to maintain paralysis.<sup>2</sup> However, as little as 25 mg in adults<sup>1</sup> and 10 mg in a patient whose weight was not given (possibly a 10-year-old child)<sup>3</sup> has been reported as sufficient to cause this effect. We suggest, however, that significantly smaller doses of suxamethonium may cause this effect. The deadspace of a 22-G Venflon<sup>4</sup> and a 25-G Butterfly is about 0.1 ml. The dose of suxamethonium given as a 'second dose' with the

flush was, therefore, probably less than 5 mg (0.3–0.4 mg/kg in the 3-year-old and only about 0.1 mg/kg in the other children). Therefore, the minimum 'second dose' of suxamethonium required to cause profound bradyarrhythmia or asystole may be even less than this.

This may be a complication that has become apparent with the use of modern monitoring. It is possible that brief periods of bradycardia or even asystole have been missed previously because the pulse has been monitored with an artefact-prone ECG lacking an alarm system. Therefore, we recommend that a pulse oximeter or an ECG (with suitably set alarms) be used routinely, no matter how short the procedure. All injections of intravenous drugs should be flushed through with normal saline to ensure none is retained in the deadspace of the cannula. This report provides a strong argument for the routine use of anticholinergic drugs at premedication or induction.

Royal United Hospital,  
Bath BA1 3NG

M.E. WILSON  
A.H. MAYOR

#### References

1. LUPPRIAN KG, CHURCHILL-DAVIDSON HC. Effect of suxamethonium on cardiac rhythm. *British Medical Journal* 1960; ii: 1774–7.
2. MATHIAS JA, EVANS-PROSSER CD, CHURCHILL-DAVIDSON HC. The role of the non-depolarising drugs in the prevention of suxamethonium bradycardia. *British Journal of Anaesthesia* 1970; 42: 609–13.
3. BULLOUGH J. Intermittent suxamethonium injection. *British Medical Journal* 1959; i: 786–7.
4. MACFIE AG. Equipment deadspace in drug administration. *Anaesthesia* 1990; 45: 145–7.

#### Fixation of nasotracheal and nasogastric tubes

Like Dr Srivatsa (*Anaesthesia* 1991; 46: 153–4), I have encountered the need for firm fixation of tubes and would like to offer an alternative method which is particularly useful in oesophageal or pharyngeal surgery.

One metre of strong thread is required; button thread or similar multifilament thread is preferred. Using a strong suction apparatus, half of the thread is offered to a soft suction catheter and is sucked through it while the other half is retained in the hand. The catheter is then passed through either nostril to the nasopharynx, one thread within and the other without. At the nasopharynx the tip of the catheter is grasped with Magills forceps, the outer thread is pulled out through the mouth and the catheter is removed leaving the inner thread from nostril to nasopharynx. The catheter is immediately inserted down the other nostril, suction is again applied and the thread in hand is sucked through the catheter which is again

removed. This leaves one thread in each nostril passing round the vomer. The uvula must be examined to ensure that it is not ensnared. The two ends of the thread are now tied together using a nonslip knot, close to the nasal columella but exerting no pressure on it. Nasogastric, nasotracheal or other tubes can now be tied by the ends of the threads as appropriate.

Thus far, no patient has been sufficiently determined as to remove a tube and no adverse sequelae have resulted. A clear note must be made instructing that only one side of the knot be cut when the thread is withdrawn.

Old Forewood Lane,  
Crowhurst,  
Battle,  
East Sussex

H. MIDDLETON

#### Pulmonary artery catheter entrapment

Difficulty with inserting a pulmonary artery (PA) catheter is a common occurrence but failure to remove one is extremely rare.

A 40 kg, 73-year-old woman had a PA catheter inserted through the left internal jugular vein under general anaesthesia for mitral valve replacement surgery. She had had a previous mitral valvotomy and mitral valve replacement, and was known to have pulmonary hypertension and kyphosis. The operation and initial postoperative course

were uneventful. PA pressure traces were useful preoperatively, but when a 'wedge' pressure could not be recorded postoperatively, the right atrial pressure was used instead.

The next morning it proved impossible to remove the PA catheter with gentle traction. When the balloon was re-inflated and then aspirated, blood was withdrawn indicating balloon rupture. A chest X ray was performed (Fig. 1), which indicated tethering of the catheter within the right atrium. Further traction led to catheter breakage leaving



# HANDBOOK OF **Obstetric Anaesthesia**

**A. S. Buchan  
G. H. Sharwood - Smith**

This concise pocket book provides:

- *junior anaesthetists with a handy revision source for post graduate examinations*
- *qualified anaesthetists with a practical reference to the management of anaesthesia in obstetric emergencies*
- *obstetricians and midwives with an excellent resumé of obstetric anaesthesia including adult and neonatal resuscitation*

Carefully selected reading lists are provided at the end of each chapter and guidelines given for the immediate management of obstetric emergencies, for example:

- *major haemorrhage*
- *failed intubation*
- *eclampsia*
- *neonatal resuscitation*

Also covered are the management of medical problems in labour, the conduct of spinal and epidural analgesia and advice on the prevention and treatment of complications.

**A S Buchan MBChB FFARCS and G H Sharwood-Smith MBChB FFARCS** are Consultant Anaesthetists at the Simpson Memorial Maternity Pavillion, Royal Infirmary of Edinburgh, UK.

0 7020 1540 7   April 1991   £9.95   128pp   6 ills   Pb

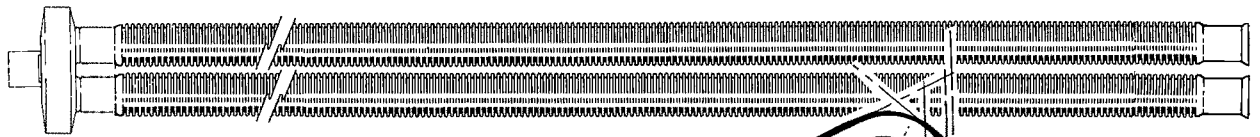
## **W. B. Saunders**

Harcourt Brace Jovanovich, 24-28 Oval Road, London NW1 7DX.

HSC/0491/A105

# The safer alternative

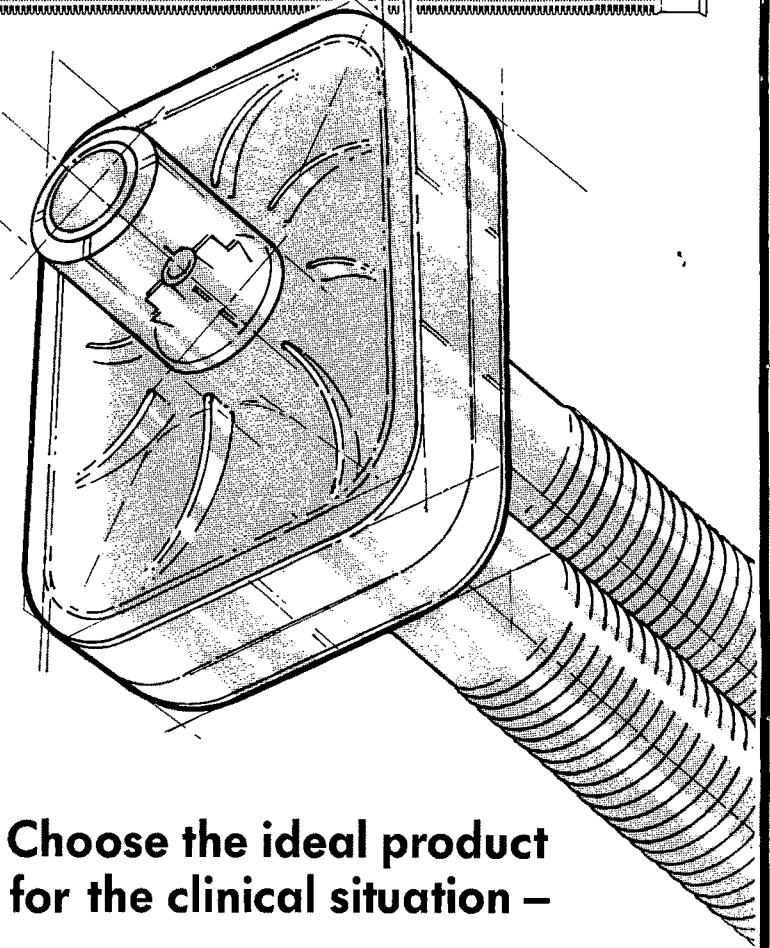
## Filtration Technology at its finest



**A range of unique Breathing Filters/HME's to resolve all your filtration and humidification problems**

The Filta-Therm – providing both Heat and Moisture Exchange and Bacterial/Viral Filtration 99.999% – ideal for **patient protection and humidification**.

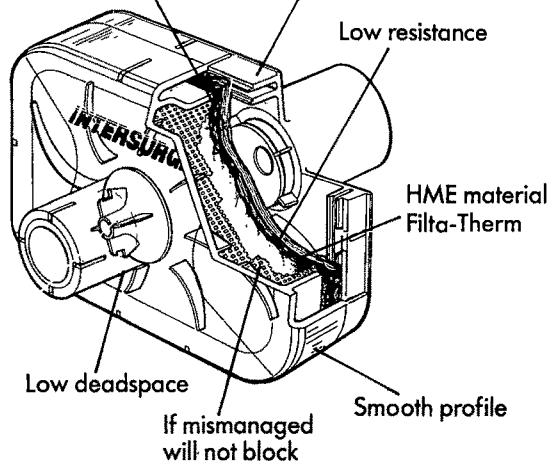
The Filta-Guard – providing Bacterial/Viral Filtration 99.999% – ideal for **equipment protection – no wastage**.



Filter matrix  
99.999% efficiency

Light-weight

Low resistance



**Choose the ideal product for the clinical situation –**

**Choose a safe product – choose Intersurgical.**

**INTERSURGICAL**  
COMPLETE RESPIRATORY SYSTEMS

Crane House, Gould Road,  
Twickenham, Middlesex TW2 6RS.  
Tel: 081-755 1477 Fax: 081-755 2177



G.M.P. Registered  
No. RO252/ME

I am interested in further details of Filta-Guard ☐ Filta-Therm ☐  
Intersurgical, Crane House, Gould Road, Twickenham, Middlesex TW2 6RS.

Name \_\_\_\_\_ Position \_\_\_\_\_ Dept. \_\_\_\_\_  
Hospital \_\_\_\_\_ Hospital Address \_\_\_\_\_

Equipment used \_\_\_\_\_

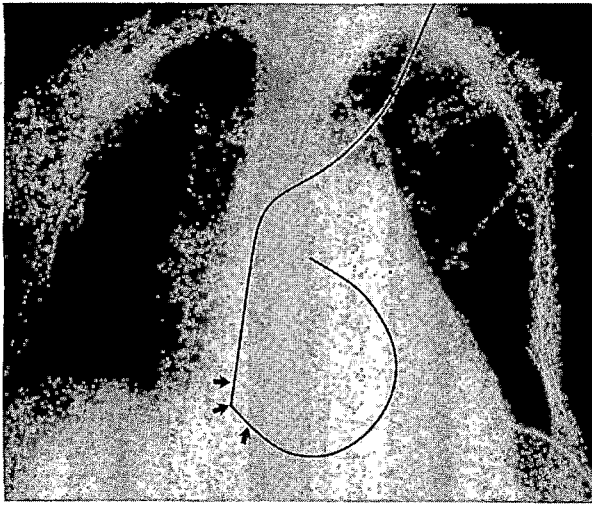


Fig. 1. PA catheter entrapment. Arrows show catheter snared at atrial ring. Note also central line, ECG leads, mitral valve and tracheal tube *in situ*.

15 cm of catheter in the patient. She was returned to the operating theatre and the distal catheter removed antero-gradely through a pulmonary arteriotomy. She made an uneventful recovery.

Complications reported in the original description of a PA catheter for clinical use included balloon rupture, thrombosis, pulmonary embolism, arrhythmias and failure

to reach the pulmonary artery.<sup>1</sup> Larger prospective studies have since demonstrated that minor complications are common, but major ones are rare,<sup>2</sup> but the subject is still contentious.<sup>3</sup> Knotting of the catheter may prevent pressure recording but seldom precludes removal of the catheter through the original venotomy, provided the balloon is deflated. In a prospective study of 6245 patients performed over 5.5 years, no complications were observed during the removal of PA catheters.<sup>2</sup> Nevertheless, in one patient the authors observed the disappearance of the PA pressure trace on tightening a purse string suture following the peroperative removal of the right atrial venous cannula.<sup>2</sup> We repeat their advice that 'the PA catheter should be moved back and forth following decannulation and repair of the heart to confirm that it is free and not caught by any sutures'.

London Chest Hospital,  
London E2 9JX

C. LANIGAN  
E. CORNWELL

#### References

1. SWAN HJ, GANZ W, FORRESTER J, MARCUS H, DIAMOND G, CHONETTE D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *New England Journal of Medicine* 1970; **283**: 447-51.
2. SHAH KB, RAO TL, LAUGHLIN S, EL-ETR A. A review of pulmonary artery catheterization in 6245 patients. *Anesthesiology* 1984; **61**: 271-4.
3. ROBIN ED. Death by pulmonary artery flow-directed catheter. *Chest* 1987; **92**: 727-31.

#### Central venous cannulation: a complication

The letter from Dr Paul (*Anaesthesia* 1990; **45**: 998-9) describing separation of the side arm from a pulmonary artery catheter introducer sheath prompts us to report a similar incident in which the superior vena cava was accidentally opened to air through a damaged central venous catheter.

A 57-year-old man was recovering from severe thoracic and spinal injuries on our neurosurgical intensive care unit. At the time of the incident he was breathing spontaneously through a tracheostomy with his bed tilted 15° head up.

He had a right subclavian triple lumen central venous catheter (Viggo Hydrocath) with the tip in the superior vena cava, sutured to the skin through its wings in the conventional manner and covered with a clear occlusive dressing. A fluid administration set was attached to one of

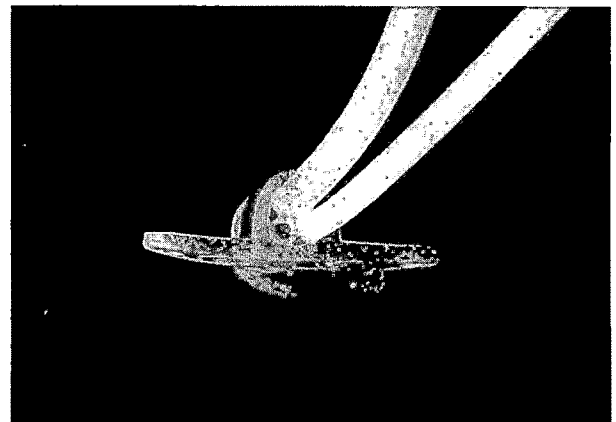


Fig. 2.

the three lumens with a Luer lock device. A trolley carrying monitoring and recording equipment was moved from the bedside and the fluid administration set connected to the central line became entangled in one wheel. The resulting tension completely avulsed the lumen to which this set was attached (Figs 1 and 2).

Although spontaneous ventilation and head-up tilt increased the potential risk of air embolus in our patient, the incident was immediately noticed by the nursing staff and appropriate action taken. Presumably the lumen was exposed to very high tension, although it is interesting that it avulsed instead of the sutures tearing.

Royal Infirmary,  
Edinburgh EH3 9YW

C.P.J. MORTON  
N.M. DEARDEN

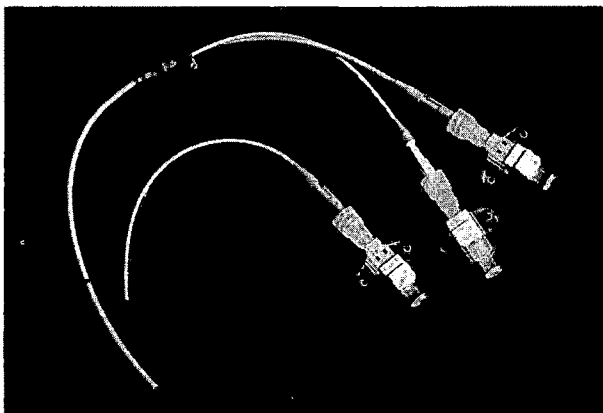


Fig. 1.

*A reply*

In the absence of having the opportunity to examine the catheter it is not possible for us to make any comments about this specific incident. It is clear that even the most robust manufacture is unlikely to be able to withstand the tensile stresses described in Dr Morton's letter. The Hydrocath central venous catheter described in this incident was manufactured to a specification which exceeds the British Standard for tensile failure of 15 Newtons. These catheters were manufactured to a minimum specification of

20 Newtons for tensile failure. Subsequently, the production process has changed to enhance manufacturing flexibility and improve the functionality of the external lumena by introducing transparent tubing. The incident described by Dr Morton serves to illustrate the importance of careful management of intravenous lines, especially those placed in the central venous system where the danger of air embolus is a persistent potential hazard.

*Viggo-Spectramed,  
Swindon SN3 5HQ*

J.P. WILKINSON

**Freezing of thiopentone solution**

Dr Thickett (*Anaesthesia* 1991; 46: 74) reported solidification of a 2.5% solution of thiopentone following storage in a refrigerator for 12 hours and suggested that perhaps this was precipitation of thiopentone acid. I am reporting a similar incident and would propose that this was due to freezing of the thiopentone solution.

General anaesthesia was required for an elective Caesarean section and drugs which had been prepared the previous day and stored in a refrigerator since then were used. Thiopentone was given to induce anaesthesia and after 10 ml had been injected it was impossible to inject any more solution. As with Dr Thickett's case, anaesthesia was induced and the rest of the anaesthetic proceeded without incident. Upon inspection the thiopentone solution was seen to have frozen and over the next few minutes it

thawed. The drugs used were stored on the top shelf of a drug refrigerator, close to the ice box which was crusted with ice, but the thermometer registered in the safe zone. Since then I have measured the temperature of the air around the ice box of a fridge in a similar condition and found it to be between  $-1^{\circ}\text{C}$  and  $-2^{\circ}\text{C}$ .

The freezing point of a 2.5% solution of thiopentone is  $-0.38^{\circ}\text{C}$ , well above the recorded temperature and this may well explain the events which happened. It would be wise not to store drugs close to the ice box of such a refrigerator in order to avoid this problem.

*Edgware General Hospital,  
Edgware,  
Middlesex*

M.H. CROSS

**The anaesthetic management of tracheal stenosis**

I read with interest the article by Drs Murphy and Lloyd-Thomas (*Anaesthesia* 1991; 46: 106-9). Since the appointment in this hospital in 1984 of a surgeon interested in upper airway problems of newborn and premature infants, I have had to address similar problems. The technique I have developed for endoscopy combines topical and general anaesthesia, as does the one described. In addition, my technique uses insufflation. This allows the surgeon a relatively leisurely examination and I believe it could be useful in 'stovepipe trachea'.

The dose (3 mg/kg) of 4% lignocaine is calculated and drawn up in a 2 ml syringe. If required it is diluted with sterile water to a manageable volume. A 24 swg Verhoeven otologic suction tube (Fig. 1) is attached. After anaesthesia is induced with oxygen and halothane, a small amount of the lignocaine is deposited in the laryngeal ventricles. Desaturation has not proved a problem even if laryngospasm occurs. Oxygen and halothane are continued for a further 2 minutes, by which time it is easy to spray the rest of the larynx and trachea. A 6 FG suction catheter is passed nasally. When its tip is level with the larynx, a mark is made on it 2-3 cm from the tip of the nose. After passing it into the trachea it is taped to the nose at the level of the mark. Anaesthesia is then maintained by insufflation of 0.5 litres/minute oxygen with halothane. A Guedel airway is used until the surgeon is ready.

I have found this technique to work well in subglottic

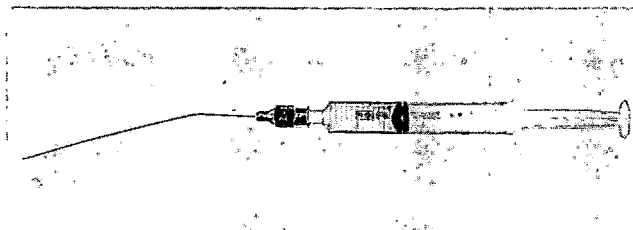


Fig. 1. Verhoeven suction tube.

stenosis and in laryngomalacia. An additional advantage to the surgeon is that during waking he can observe vocal cord mobility. In calibrating the trachea it is useful to remember that the external diameter of the catheter is almost exactly 2 mm. A similar technique has been described in an otolaryngology paper,<sup>1</sup> but not to my knowledge in an anaesthetic journal.

*Ninewells Hospital and  
Medical School,  
Dundee DD1 9SY*

I.G. GROVE-WHITE

*Reference*

1. BENJAMIN B. Anaesthesia for laryngoscopy. *Annals of Otolaryngology and Rhinology* 1984; 93: 338-42.

**Contiplex (Braun) Katheterset**

We wish to draw to the attention of anaesthetists who are considering ordering the Contiplex (Braun) Katheterset for continuous plexus block, the following anomaly. We were mystified by the English instructions urging us to advance the introducer needle 'until red plastic connector hub has reached the skin level'. There was nothing remotely red in the set and no reference to it in the German instructions. Thus, thanks to a German presence in our department, we are negotiating a discount!

*Ealing Hospital,  
Southall,  
Middlesex UB1 3HW*

G.A. FRITZ  
E.G. BRADSHAW

*A reply*

The Contiplex set and the instructions for use seem to refer to a batch manufactured in 1989. We realised the incorrect statement in our instructions for use and made appropriate corrections in 1990. We apologise for any confusion and inconvenience caused.

*B. Braun Melsungen AG,  
Medical Products Division,  
P.O.B. 110 + 120,  
3508 Melsungen, Germany*

H.J. OTTO

## Obituaries

- Walton, W.J., MB, BS, DA, FFARCS, FFARCSI.** Formerly Consultant Anaesthetist at Derbyshire Royal Infirmary. Qualified from University of London in 1956.
- Griffiths, H.W.C., FRCSEd, FFARCS.** Formerly Consultant Anaesthetist at Edinburgh Royal Infirmary. Qualified from Calcutta in 1941.
- Lang, P.V., MB, BCh, BAO, FFARCSI.** Formerly Consultant Anaesthetist at Altnagelvin Hospital, Londonderry. Qualified from University College, Dublin 1940.
- Warren, S.J., MB, BS, FFARCS.** Formerly Consultant Anaesthetist, Freeman Hospital, Newcastle. Qualified from University of London 1978.
- Dorrian, G., FFARCSI.** Formerly Consultant Anaesthetist at St. Vincent's Hospital Dublin. Qualified from University College, Galway, 1976.
- Hunter, A.R., MD, FRCS(Glas) FFARCS.** Formerly Professor of Anaesthesia, University of Manchester. Qualified from University of Glasgow in 1937.

## International congress calendar

### 1991

- 21–23 August.** Edinburgh. *Edinburgh Anaesthesia Festival.*  
Information: Dr A.J. Pollock, Department of Anaesthetics, Royal Infirmary, Edinburgh EH3 9YW.
- 22–24 August.** Auckland. *The Annual Conference of New Zealand Anaesthetists.*  
Information: Department of Anaesthesia, Auckland Hospital, Park Road, Auckland, New Zealand.
- 25–31 August.** Willemstad, Curacao, Netherlands Antilles. *19th Meeting of the International Society of Oxygen Transport to Tissue (ISOTT).*  
Information: Mrs Denise Haas, Department of Anaesthesiology, Erasmus University, Dr Molewaterplein 40, 3015 GD Rotterdam, The Netherlands.
- 28 August–1 September.** Strasbourg. *European Academy Scientific Meeting.*  
Information: Professor J.C. Otteni, Service d'anesthésie et de Réanimation, Hôpital de Hautepierre, Avenue Molière, F-67098 Strasbourg, France.
- 1–3 September.** Oxford. *British Sleep Society, 3rd Annual Meeting.*  
Information: Dr J. Stradling, Osler Chest Unit, Churchill Hospital, Headington, Oxford OX3 7LJ.
- 1–7 September.** Paris. *1st World Congress of Cellular and Molecular Biology.*  
Information: Mrs Leila Orbecchi, Director, C.E.R.T., 63 Avenue Parmentier 75 011 Paris, France.
- 4–8 September.** Rio de Janeiro. *XXI Latin American Congress of Anaesthesiology (WFSA).*  
Information: Dra M.B. de Azeveda, Rua Paulo Barreto 60, Botafogo, CEP 22280 Botafogo, Rio de Janeiro, RJ, Brazil.
- 6 September.** Bordeaux, France. *Third International Workshop on High Frequency Jet Ventilation.*  
Information: Dr A.M. Cros, Département Anesthésie — Réanimation IV, Hôpital Pellegrin, Place Amélie Raba-Leon, 33076 Bordeaux Cedex, France.
- 6–8 September.** Texas. *Texas Society of Anesthesiologists.*  
Information: Texas Society of Anesthesiologists, 1905 North Lamar Boulevard, # 107, Austin, Texas 78705, USA.
- 11–13 September.** Harrogate. *Linkman and Annual Scientific Meeting of Association of Anaesthetists of Great Britain and Ireland.*  
Information: Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 17–21 September.** Patras, Greece. *9th Greek Congress of Anaesthesiology, Intensive Care and Emergency Medicine.*  
Information: Prof. C. Alexopoulos, Department of Anaesthesiology, Medical School, King George Square 30, Patras 262 21-Greece.
- 21–23 September.** Tokyo, Japan. *Third World Congress on Sleep Apnea and Rhonchopathy (III WCSAR).*  
Information: Secretariat for III WCSAR, Simul International Inc., Kowa Building, No. 9, 1-8-10 Akasaka, Minato-ku, Tokyo 107, Japan.
- 26–27 September.** Bordeaux France. *Third Course on high frequency jet ventilation (HFJV).*  
Information: Dr A.M. Cros, Hôpital Pellegrin-Tripode Level 9, Place Amélie Raba-Leon, 33076 Bordeaux Cedex, France.
- 11–15 October.** Baghdad. *4th Pan-Arab Congress of Anaesthesia and Intensive Care.*  
Information: Dr M. Keilani, P.O. Box 17078, Amman, Jordan.
- 26–30 October.** San Francisco. *American Society of Anesthesiologists Annual Meeting.*  
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 6–9 November.** Kuala Lumpur. *7th Asian Congress of Anaesthesiologists.*  
Information: Dr S.W. Lim, Pantai Medical Centre, 59199 Kuala Lumpur, Malaysia.
- 8–10 November.** Vina del Mar. *2nd Congress of Fed. of South American Soc. of Anesthesiologists.*  
Information: Dr Guillermo Lema, Av. Providencia 1476 (Depto. 405) Santiago, Chile.
- 8–11 November.** Toronto. *Paediatric Anaesthesia Conference.*  
Information: Sheila M. Peart, Paediatric Anaesthesia Conference, The Hospital for Sick Children, 555 University Avenue, Toronto, Ont M5G 1X8.
- 13–15 November.** Beer-Sheva, Israel. *Fifth International Symposium on Anesthesia and Intensive Care.*  
Information: Dr G. Gurman, Division of Anaesthesiology, Soroka Medical Centre, Beer-Sheva 84101, Israel.
- 1–4 December.** Bangkok. *6th Congress of Western Pacific Association of Critical Care Medicine.*  
Information: Dr P. Sakolsatayadorn, Surgery, Siriraj Hospital, Bangkok 10700, Thailand.
- 6–8 December.** Washington. *Washington State Society of Anesthesiologists Annual Meeting.*  
Information: Washington State Society of Anesthesiologists, 2033 Sixth Avenue, # 804, Seattle, Washington 98121, USA.
- 7–11 December.** New York. *Forty-fifth Postgraduate Assembly in Anesthesiology.*  
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

### 1992

- 1–8 February.** Colorado. *18th Annual Vail Conference in Anaesthesiology.*  
Information: Sonja Craythorne, Professional Seminars, P.O. Box 012318, Miami, Florida 33101, USA.
- 29 February–1 March.** Lubbock, Texas, USA. *Texas Tech University Health Sciences Center's Annual Refresher Course in Anesthesiology.*  
Information: Barbara L. Smith, Department of Anesthesiology, 3601 4th Street, Room IC-282, Lubbock, Texas 79430, USA.
- 13–17 March.** San Francisco. *66th Congress of the International Anesthesia Research Society.*  
Information: International Anesthesia Research Society, 3645 Warrenville Center Road, Cleveland, Ohio 44122, USA.
- 16–19 March.** Johannesburg, South Africa. *South African Society of Anaesthetists Congress.*  
Information: Professor D.F. Morrell, Area 361, Johannesburg Hospital, Private Bag X39, Johannesburg 2000, South Africa.

- 25-29 March.** Tampa. *17th Annual Meeting of the American Society of Regional Anesthesia.*  
*Information:* P.O. Box 11086, Richmond, Virginia, 23230-1086, USA.
- 26-28 March.** Martinique, French West Indies. *Second International Meeting of Anaesthesia and Intensive Care.*  
*Information:* Congres SMAAR, Departement d'Anesthesie Centre Hospitalier, 97232 Le Lamentin, Martinique, F.W.I.
- 29 March-2 April.** Atlanta, Georgia. *The Third International Symposium on the History of Anaesthesia.*  
*Information:* R.K. Calverley, Medical Center, University of California, 225 Dickinson Street, San Diego, California CA 92103, USA.
- 1-3 April.** Bristol. *Junior Anaesthetists' Group Linkman Conference and Annual Scientific Meeting.*  
*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 9-11 April.** Fukuoka, Japan. *39th Annual Meeting of Japan Society of Anaesthesiology.*  
*Information:* Kenjiro Dan, Department of Anesthesiology School of Medicine, Fukuoka University, 45-1, 7-chome Nanakuma, Jonan-ku, Fukuoka, 814-01, Japan.
- 23-25 April.** Atlanta, Georgia. *Second International Symposium on Memory and Awareness in Anaesthesia.*  
*Information:* Susan J. Duensing, Continuing Medical Education Programme Director, Emory University School of Medicine, 1440 Clifton Road NE, 109 WHSCAB, Atlanta, Georgia 30322, USA.
- 2-6 May.** Boston. *Society of Cardiovascular Anesthetists.*  
*Information:* P.O. Box 11086, Richmond, Virginia, 23230-1086, USA.
- 4-9 June.** Toronto. *49th Annual Meeting of the Canadian Anaesthetists' Society.*  
*Information:* 187 Gerrard Street E, Toronto, Canada M5A 2E5.
- 7-12 June.** Barcelona. *Anesthesia 92.*  
*Information:* Pacifico, S.A.: c/Muntaner, 112 08036-Barcelona, Spain.
- 9-12 June.** Haifa, Israel. *16th International Congress of the Israel Society of Anaesthesiologists.*  
*Information:* Secretariat, Anesthesiologists, PO Box 50006, 61500 Tel Aviv, Israel.
- 10-13 June.** Brussels. *European Society of Regional Anaesthesia (UK) Meeting.*  
*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 12-19 June.** The Hague. *10th World Congress of Anaesthesiology.*  
*Information:* Dr Harm Lip, Nilantweg, 99, 8041 AR Zwolle, Netherlands.
- 9-11 September.** Bournemouth. *Linkman and Annual Scientific Meeting.*  
*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 13-18 September.** Jerusalem, Israel. *5th International Congress of the Pain Clinic.*  
*Information:* 5th International Congress, The Pain Clinic, PO Box 50006, 61500 Tel Aviv, Israel.

- 17-21 October.** New Orleans. *American Society of Anesthesiologists Annual Meeting.*  
*Information:* Executive Secretary, ASA, 515 Busse Highway, Park Ridge, IL 60068, USA.
- 12-16 December.** New York. *46th Postgraduate Assembly in Anesthesiology.*  
*Information:* Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists, Inc., 41 East 42nd Street, Suite 1605, New York 10017, USA.

# 1993

- 12-16 February.** Utah. *38th Annual Postgraduate Course in Anesthesiology — 'Anesthesiology: Today and Tomorrow'.*  
*Information:* Vicky Larson, Department of Anesthesiology, University of Utah School of Medicine, 50 North Medical Drive, Salt Lake City, Utah 84132, USA.
- 12-15 March.** Hong Kong. *7th Congress of Western Pacific Association of Critical Care Medicine.*  
*Information:* Ms Elma Lindsay, International Conference Consultants Ltd., 1st Floor, 57 Wyndham Street, Central, Hong Kong.
- 29 April-2 March.** North Carolina. *Meeting of the Association of University Anesthetists.*  
*Information:* Francis M. James III, Department of Anesthesia, Wake Forest University Medical Center, 300 S. Hawthorne Road, Winston-Salem, North Carolina 27103, USA.
- 1-4 September.** Liverpool. *European Course and Congress in Paediatric Anaesthesia.*  
*Information:* Dr P.D. Booker, Alder Hey Hospital, Liverpool L12 2AP.
- 22-24 September.** Glasgow. *Linkman Conference and Annual Scientific Meeting.* Joint Meeting between the Association of Anaesthetists of Great Britain and Ireland and the Canadian Anaesthetists' Society.  
*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 9-13 October.** Washington DC. *American Society of Anesthesiologists Annual Meeting.*  
*Information:* Executive Secretary, ASA 515 Busse Highway, Park Ridge, IL 60068, USA.

# 1994

- 7-9 September.** Brighton. *Linkman Conference and Annual Scientific Meeting.*  
*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.
- 2-7 October.** Jerusalem. *European Congress of Anaesthesiology.*  
*Information:* The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London, WC1B 3RA.



## The Annual Conference of Linkmen of the Association of Anaesthetists of Great Britain and Ireland, 1990

The Honorary Treasurer, Dr W. R. MacRae welcomed 236 Linkmen to this meeting, held at the University of Manchester Institute of Science and Technology (UMIST), on 26 September 1990. He informed Linkmen of the recent illness of the President Dr M. M. Burrows. At the same time he noted that Dr Burrows was making good progress and had expressed regret that he was unable to attend. Dr MacRae spoke for everyone when he wished Dr Burrows a speedy and complete recovery.

He welcomed all present and emphasised that the Linkman conference was one of the Association's most important events. The exchange of views between members and the Council at last years' meeting had led to many new initiatives on the part of the Association, which would be reported to the meeting. He stressed the need for continuing dialogue within the Association to permit the correct expression of members' views.

### Anaphylactic reactions associated with anaesthesia

Dr W. S. Nimmo (*Edinburgh*) began by explaining that the Association had been requested to give advice on this topic following a fatal accident enquiry in Aberdeen. A female patient had died from a massive anaphylactoid reaction which occurred at the induction of anaesthesia. The Sheriff conducting the enquiry had recommended that the introduction of routine screening tests for patients presenting for elective surgery should be considered. This suggestion received considerable publicity and members of the Association had requested guidance. Accordingly, a working party had been formed to examine this matter consisting of Professor A. R. Aitkenhead (Nottingham), Professor R. S. J. Clarke (Belfast), Dr R. M. Weller (Bristol) and himself as Chairman, (Officers, ex officio).

The objectives of the working party were to review anaphylactic reactions associated with anaesthesia, to consider recognition and treatment of these reactions, to make recommendations about the treatment of reactions and to consider the role of screening for anaphylactic reactions before anaesthesia.

Dr Nimmo reviewed definitions used by the working party and the likely incidence of severe anaphylactic reactions. He noted the rarity of this event and suggested that even where this occurred in association with anaesthesia, a prompt and successful recovery could be anticipated with appropriate treatment. He emphasised that every doctor who gives drugs must be able to recognise and treat such reactions.

The introduction of an 'anaphylaxis drill' was an important part of the management. It should be agreed by all departments as a standard operating procedure and be available in all anaesthetising locations. This drill should be rehearsed at regular intervals and all members of staff involved should be familiar with it. The use of intravenous adrenaline at an early stage was commended, particularly in the presence of bronchospasm.

Dr Nimmo said that any patient suspected of having an anaphylactic reaction should be fully investigated, that the result should be made known to the patient and that the reaction should be reported to the Committee on the Safety

of Medicines. He emphasised that it is the responsibility of the anaesthetist to see these things are carried out, and that this cannot be delegated. In addition, prospective arrangements for investigation of suspected anaphylactic reactions should be made with an immunology department. He reviewed the sort of testing that might be done, but stressed that there are no tests which can be carried out at the time of a reaction that have been shown to provide useful information for immediate clinical management. Finally, he noted that there is no support for routine screening of patients for specific drug antibodies prior to anaesthesia at present, nor are there any predictors of anaphylaxis.

### Discussion

Dr S. R. Swindells (*Leeds*) enquired about crossover reactions with antibiotics. Dr Nimmo professed that he had an intense dislike of the routine use of prophylactic drugs, particularly when given intravenously and when prescribed by someone else. He felt that such drugs were given frequently at an inappropriate time and that the best solution was for the surgeon requiring the drug to be given, to give it personally and at the correct time. Dr J. W. Martin (*Burton*) questioned the need for regular practice drills and felt that this was something that all anaesthetists should know. Dr Nimmo felt that it was reasonable to carry these out. Dr R. P. Harpin (*Newcastle upon Tyne*) noted that the working party had stopped short of defining drugs most likely to cause problems. In reply Dr Nimmo stated that he felt the data were not hard enough to make statements about specific agents.

Dr D. J. Elliott (*Ipswich*) enquired whether there was a relationship between the anxious, agitated patient and the likelihood of anaphylaxis. Dr Nimmo felt this was possible, but that there were no supportive data. Dr M. E. Ward (*Oxford*) criticised the layout of the Association booklet. He felt that undue prominence had been given to the Sheriff's comments without adequate rebuttal. He was concerned that trainees might get the wrong impression. In reply, Dr Nimmo noted this concern, pointing out that the summary came first in the booklet, thus ensuring that all the recommendations received equal prominence. He conceded there may be a problem if the document fell into lay hands. Dr R. L. Hargrove (*Westminster*) expressed concern about the medicolegal consequences of a statement in the booklet and illustrated his concern with an example.

### Department of Health Working Party on dental anaesthesia

Dr D. A. Zideman (*Hammersmith*) outlined events leading to the formation of the expert working party, informed Linkmen of its composition and terms of reference and reviewed previous working party reports on the same topic. He noted that the report was applicable only to England and not to other parts of the United Kingdom. The working party was to make recommendations concerning safe practice and training in general anaesthesia and sedation in dentistry. Dr Zideman presented data showing the numbers of general anaesthetics and patients who received

sedation. He noted that the figures quoted did not include those from the private sector.

The working party had held over a dozen meetings and received over 100 submissions of evidence. He understood that the document would be published as a consultative document and circulated to the estimated 250 bodies thought to have an interest. Because the report had not been published, and thus remained confidential, Dr Zideman was unable to give more than a flavour of the report. He felt that if adopted, the recommendations were good, and would have a positive influence for the safety of patients receiving general anaesthesia or sedation for dentistry.

### Discussion

Dr D. J. Lightman (*Harlow*) informed the meeting that he no longer gave general dental anaesthesia outside the hospital environment as he could not provide what he believed was the requisite standard of care elsewhere. He wondered whether the Association should recommend that general anaesthesia should not be given outside hospital. Dr Zideman answered by saying that the report may reflect some of Dr Lightman's concerns. Dr MacRae noted that the Association had made very strong representations to the working party. Dr D. S. Arthur (*Glasgow*) asked if anaesthesia for dentistry could be restricted to properly trained anaesthetists, or alternatively that the dentists' right to give general anaesthesia might be restricted. Dr Zideman outlined the problems associated with this approach and speculated why no one was advocating the wider use of sedation as an alternative.

Dr K. J. Gill (*West Dorset*) commented upon the poor take up of places on courses providing resuscitation training. Dr Zideman noted that improved training was likely to be a recommended requirement of the working party and he anticipated that all staff concerned with the administration of general anaesthesia and sedation would need to attend. Dr E. B. Lewis (*Hythe*) suggested that the poor level of fees for dental anaesthesia might be responsible for the decline in general anaesthesia in the NHS, and that a commensurate increase in activity in private practice might be the result. He noted that many working party reports tried to impose rapid introduction of their recommendations. He suggested that a 10-year phased introduction for the recommendations might ensure a higher success rate.

Dr Stephanie Greenwell (*North Tyneside*) informed the meeting of the closure of local clinics which could not reach safety standards. These had been accommodated now in a purpose built facility which was running well and provided an excellent training environment. Dr M. Cody (*Enniskillen*) noted the larger numbers of patients who presented for general anaesthesia for dentistry in Northern Ireland. In reply, Dr Zideman felt that he could not comment upon the situation in Northern Ireland, but did agree with Dr Lewis' suggestions about the increase in the numbers of general dental anaesthetics in private practice.

### Safety and monitoring for endoscopy under sedation

J. E. Charlton, Assistant Honorary Secretary, outlined the gestation of a working party of the British Society of Gastroenterology (BSG). The endoscopy section of the BSG had formed a working party to consider methods of improving the safety of endoscopy under sedation. Membership of the working party was divided equally between surgeons and physicians. Dr Charlton had attended by invitation to provide an opinion about safe monitoring practice.

From the outset it was obvious that there were large differences of opinion as to what might be termed safe practice. Dr Charlton gave examples which included the use of monitoring, supplementary oxygen, training and availability of assistance, intravenous access and postendoscopy care. He felt that the final report was unlikely to be perfect, but represented about as much as it was possible to achieve at present. He was pleased to note that the vast majority of the members of the working party had changed their own practice as a result of their being a member. The final report of the working party would be submitted to the Council of the BSG for approval. If this was obtained the report would be published in GUT. It was intended to make further comment through the correspondence columns of GUT at the time of publication.

### Discussion

Dr P. A. Wilson (*Kilmarnock*) drew attention to the problem of anaesthetising patients following emergency endoscopy, when high dosage of intravenous benzodiazepines had been administered. Dr D. A. Wilkinson (*St Bartholomew's Hospital*) alluded to the hazards of high doses of intravenous opioids and topical lignocaine during fibre optic bronchoscopy performed by physicians.

Several Linkmen emphasised the need for endoscopists to be trained in resuscitation techniques and the use of intravenous sedation. It was hoped that this Report would convince them of this need. Dr Charlton urged anaesthetists to play an active role if requested to provide such training. It was suggested that there should be a legal requirement for endoscopists to be proficient in resuscitation. Dr Margaret Heath (*Lewisham*), felt that anaesthetists should not manufacture rods for their backs. She suggested that ODAs, trained by anaesthetists, could provide skilled assistance during endoscopy.

### Honorary Secretary's update

Dr W. L. M. Baird, Honorary Secretary, reported that interest in the affairs of the Association has never been stronger, as evidenced by the fact that no less than 14 superb candidates had offered themselves for election to Council. The Association has continued its policy of producing reports on matters of concern and interest to members in the form of glossy booklets. The Reports published include *Workload for consultant anaesthetists*, *Anaphylactic reactions associated with anaesthesia* and *Checklist for anaesthetic machines—a recommended procedure based on the use of an oxygen analyser*. The possibility of producing plasticised copies of excerpts from the latter two documents, to be available in the operating department, was being investigated. The production of two other documents was at an advanced stage; the *Careers guide* was with Ohmeda for the addition of illustrations and the *Handbook for trainee anaesthetists*, formerly the SHO Handbook, was nearing completion.

Revised guidance to members applying for Association funding was now available in Educational Awards. This document had been prepared by successive Chairmen of the Education and Research committee, Dr W. S. Nimmo and Dr J. A. W. Wildsmith with the assistance of Dr J. F. Nunn. Six applications were received for the 1990 Association Research Fellowship, but after interviews it had been decided to make no appointment. The Research Fellowship would be re-advertised early in 1991. Applications were also sought for the Baxter Travelling Fellowship.

The last year has seen the continuation of the enormously successful programme of Seminars at 9 Bedford

Square. Early application is recommended to ensure a place. Council is grateful to Dr Wildsmith, Seminars Co-ordinator and members of the organising committee Dr J. F. Nunn, Surgeon Commander S. Merrill and Mrs Lesley Ogg, BOC Educational Co-ordinator. Council welcomes Professor A. R. Aitkenhead as Seminars Co-ordinator for the coming year.

Dr Baird noted that this year was the last time for 5 years that the Annual Scientific Meeting would be held on a university campus. Next year, the meeting would be held at Harrogate Conference Centre. University accommodation would not be available, but there would be a wide range of accommodation to suit all needs. The JAG Meeting had reached a new peak at the Glasgow meeting with the largest number of registrants and the largest technical exhibition ever achieved. Venues for both the Annual Scientific Meeting and the JAG Meeting were now agreed until 1995.

In December 1989 the following resolution was agreed by Council — 'Council of the Association of Anaesthetists recognises the need for the College of Anaesthetists to extend its development by seeking legal identity through a separate charter, by seeking Royal status and by the acquisition of independent premises. Council of the Association is unanimous in expressing its strong support to the College of Anaesthetists in pursuing these aims'. This resolution received unanimous support of members attending the 1990 Winter Scientific Meeting.

The annual meeting of Officers of the Association with representatives of the specialist societies had produced the idea that a session of the Winter Scientific Meeting would be run by one of the specialist societies. This would act as a 'shop window' for their specialty and provide information that would be of value to the nonspecialist. The first such session would be at the next Winter Scientific Meeting and would be organised by the Association of Paediatric Anaesthetists. Consideration was also being given to the suggestion that meetings of specialist societies could be run 'back to back' with Association meetings, and the views of Linkmen were sought.

Dr Baird reviewed the complex series of events that accompanied the introduction of new levels of benefit by the British United Provident Association (BUPA). The Association has been swamped with letters and phone calls of complaint. Attempts had been made to meet with higher executives of BUPA but these had not been forthcoming. Consequently, meetings had been arranged between Officers of the Association and representatives of BUPA. After strong pressure from the Association BUPA had agreed to raise the benefit available for all minor procedures to the level of minor 5. The Association has offered to meet again with BUPA representatives before this meeting but had been unable to convince them of the urgency of the matter. A further meeting was planned for the end of October. Dr Baird thanked members for their letters and phone calls of encouragement and urged them to continue their support.

Finally, Dr Baird thanked all who supported him during his 2 years as Honorary Secretary, especially the Linkmen, without whose interest and activity the Association could not function.

### Open forum 1

#### *College of Anaesthetists*

Professor M. Rosen, President of the College of Anaesthetists, reviewed the need for an independent charter. He noted that it was now time for a real change. The standards on audit and accreditation that have been set required a strong and independent College to ensure

that they were met. The College would give a lead in multidisciplinary areas such as intensive care and pain relief. He expressed satisfaction that the Royal College of Surgeons of England had passed a resolution in support of our need to have an independent charter. He hoped that the Privy Council might grant this privilege next year.

Professor Rosen outlined the time course towards independent status, the need to purchase a prestigious building and the sums necessary to achieve this. He emphasised the need for the support of every Fellow. He expressed the hope that the College would be granted Royal status soon after receiving the charter.

#### *Site for the College*

In discussion, Dr R. P. Harpin (*Newcastle upon Tyne*) expressed concern about the aggregation of resources in London. Professor Rosen stated that the College was running regional refresher courses and would encourage the further development of regional education. The format of the College had yet to be determined and he did not rule out regional representation. Dr P. E. Daly (*Royal Marsden*) asked why the College needed to be sited in London. In reply Professor Rosen expressed the view that this was the only practical site that could have been chosen. All the bodies they would have to deal with were there, and when combined with the population base and the national travel arrangements it meant there was no other choice. There was also the matter of attendance at meetings; London meetings attracted about twice the number of registrants than meetings held elsewhere, and he gave the recent meeting in Birmingham as an example.

#### *Fundraising for the College*

Dr M. E. Ward (*Oxford*) asked if the money collected by the Anaesthetists Academic Foundation was included in the total sum collected so far. Professor Rosen said it was. Dr D. E. Senior (*Dartford*) enquired whether there would be a repayment from the Royal College of Surgeons on the basis of those monies contributed by anaesthetists in previous years. Professor Rosen expressed the personal view that we could not expect anything. He noted that surgeons could donate both money and services to the College appeal, and that three already had. Dr Jennifer Jenkins (*Great Yarmouth*) suggested that 9 Bedford Square might be sold to help the College appeal. Professor Rosen advised Linkmen that this was not possible as the two organisations were quite separate and cannot be amalgamated. He noted that the *British Journal of Anaesthesia* had agreed to a half million pound interest free loan to help the College find suitable accommodation. The Honorary Treasurer, Dr W. R. MacRae, intimated that the Association was considering similar support.

Dr G. T. Whitfield (*Scarborough*) pressed Professor Rosen on the costs of a London based College, expressing the view that costs to those members working in peripheral districts were very considerable. Professor Rosen's view was that London was the best place, but noted that the College would seek the maintenance of proper provision of study leave and expenses. There should be no need to fight at local level as they are part of terms and conditions of service. Local variation would effect training and thus hospital recognition.

Dr Sheelagh White (*Inverness*) expressed the view that it was unrealistic to ask members living in areas with a low population and reduced economy to raise large sums of money. Professor Rosen felt that these factors would be no deterrent at all and offered to do all that he could to help. Professor Rosen urged members to seek publicity for the

activities of anaesthetists. This would help raise money for the appeal, and would also help patients to appreciate our standards. He emphasised that the future was bright, but that it could not be achieved without the whole-hearted support of all.

The Chairman offered a vote of thanks to Professor Rosen and expressed the hope that there would be a rapid and successful outcome to the College's quest for independent status.

### The conduct of independent practice

Dr R. M. Weller, the Chairman of the Private Practice Committee reviewed the events in the independent sector and outlined areas where effort had been directed, such as intensive care and epidurals. He noted the problems associated with a system of reimbursement that was based on the surgery performed.

In 1989 the British Medical Association (BMA) had produced guidelines for private practice fees, which had ignored the advice of specialist advisers in anaesthesia. The BMA recommendations were unrelated to our guidelines and were unacceptable to the Association. The Association had advised that these BMA guidelines should be ignored.

The Association's current guidelines covered a one-year period only, and on review it was apparent that many consultants charged less than the guidelines. This had set an artificial ceiling upon anaesthetic fees, the significance of which had now become apparent.

The Private Practice Committee had maintained pressure upon the provident associations to pay only accredited anaesthetists. When the General Medical Council specialist register is introduced this anomaly may be resolved but a problem may remain if nonaccredited and nonconsultant anaesthetists are allowed to continue practice in the independent sector with the support of local consultants.

In July 1990 BUPA, without warning or prior negotiation, had introduced new benefit levels which gave large increases to surgeons and physicians and effectively decreased the benefit payable to anaesthetists. BUPA claimed that the new benefit levels were based on a survey of fees charged for selected procedures. The new benefit levels ignored the previous verbal assurances of the Medical Group's Advisor of BUPA that the Association's fee guidelines were 'fair and reasonable'. Dr Weller emphasised to Linkmen that the strongest possible representations had been made to BUPA by the Association. As a result BUPA has reinstated the level of benefit available for all minor procedures to levels in accordance with the Association guidelines. However, there remain significant shortfalls on the benefit levels available for intermediate, major and more complex procedures.

Dr Weller stressed that a major part of the problem stemmed from the fact that a proportion of anaesthetists did not charge fees in accordance with Association guidelines. He said he could offer no explanation for this, but felt that only by acting together would a speedy resolution be possible. He noted that the BMA was intending to revise their fee guidelines. He had written to the BMA urging that consultations about their proposed recommendations be held as soon as possible, but had not yet received a reply. In conclusion, Dr Weller speculated that the system of Relative Values employed in the USA might be fairer to all concerned.

### Discussion

Dr A. C. Thurlow (*St George's Hospital*) felt that there were two important points at issue. Firstly, that the Association should devise a letter that would address the

problem of the shortfall, and secondly, there should be a separate scale of fees for London. Replying to the first point, the Chairman stated that a letter from the Association might raise accusations that we were trying to create a cartel and that this might attract adverse attention. The Assistant Honorary Secretary, Dr J. E. Charlton, explained that he used a pre-anaesthetic assessment form to explain to all patients the nature of the current problem. If there was a shortfall due to inadequate BUPA benefit levels the patient had been warned beforehand and was aware that any shortfall was their responsibility. Dr P. J. F. Baskett had adopted a similar procedure. He noted that to some extent the problem was of anaesthetists' own making, and that this was demonstrated by the Association guidelines forming an artificial ceiling for fees, thus limiting the natural range of fees.

Dr K. K. Dutt (*Birmingham*) outlined problems associated with determining the appropriate fee for procedures carried out for physicians. Dr Weller recommended that in this case the consultation fees recommended by the BMA would serve as a useful guide. Dr J. Freeman (*Kettering*) felt that an important principle and anaesthetist's prestige were involved and this could be addressed best by direct negotiation of fees for private patients and not merely with the insurance companies. Dr T. D. E. Sharpe (*Belfast*) stated that the credibility of anaesthesia had taken a dent and that patients, by and large, were ignorant of the risks of anaesthesia or the skills required for the safe practice of anaesthesia. Dr P. V. Scott (*Bromsgrove*) suggested that a separate charge for pre-operative consultation might solve the current problem. Dr Weller expressed his preference for the system of a comprehensive fee which has been the traditional method employed by the Association when drawing up guidelines.

Dr R. L. Hargrove (*Westminster*) told the meeting that he was an adviser to BUPA. He outlined the efforts he had made to prevent the adoption by BUPA of the new levels of benefit for anaesthesia. He and other colleagues had tried to introduce a reasonable system of classification of procedures for the purposes of assessing benefit, but this advice had been ignored. His advice to the meeting was clear and unequivocal; it was to charge the fees recommended by the Association. Dr E. B. Lewis (*Hythe*) supported Dr Hargrove. He too, advocated a concerted approach and noted that if this was adhered to, a settlement was possible. He felt that containment of professional fees was reasonable, but that this should never be achieved at the expense of a single group such as was the case now. The Chairman closed this section by urging those who charged fees for professional services at less than the rate recommended by the Association to think again; by doing so they were weakening the position of their colleagues.

Further contributions to this discussion were made by Dr D. Iyer (*Maidstone*) and Dr M. E. Ward (*Oxford*).

### Workload for consultant anaesthetists: The new contract

In introducing the document on workload for consultant anaesthetists, Dr MacRae paid tribute to the enormous contributions of the President Dr M. M. Burrows. He pointed out that this document was produced jointly by the Association and the College in what he described as a vital co-operation. The importance of the document lay in the fact that it described clearly the wide range of activities carried out by consultant anaesthetists as a normal part of their duties. On occasion, the extent of duties carried out by consultant anaesthetists had not been appreciated fully by managers or other colleagues. The document should be used when drawing up the new consultant job plans that were required by Spring 1991. In addition, the document

would be of help to REAs and others approving or drawing up job descriptions.

Dr MacRae drew attention to specific parts of the booklet. He noted that when the 10% limit for private practice earnings was breached the appropriate change should be made from a whole-time to a maximum part-time contract. He drew attention to the sample work programmes given in the workload document, and noted the difference between 'fixed sessions' and work actually done. Where contracts were to be held by one of the forthcoming trusts, it would be important for consultants to agree and be satisfied by superannuation arrangements consequent to the change in contract.

Dr N. O'Donovan (*North Devon*) raised the problems of on-call commitments in small hospitals with reduced numbers of trainees or inexperienced staff. He felt there should be a larger notional half day (NHD) allowance for on-call work under these circumstances. Dr M. Pegg (*Royal Free*) noted that most of her colleagues were doing seven theatre sessions, the equivalent of nine NHDs in theatre. The President-Elect, recommended the advice contained in the workload document that the hours worked be divided by three and a half to give a NHD equivalent and that between 5 and 7 should be designated as fixed. Any shortfall would have to be adjusted over a period of time. Dr D. M. Jackson (*Swindon*) felt that detailed consideration of the examples given in the document showed that they were in excess of contractual obligations.

Dr J. N. Hodkinson (*South Cumbria*) spoke concerning the 'consultant-only hospital'. In these circumstances on-call was in-call and he believed that these sessions should be regarded as fixed. He felt that, if the amount of in-call normally done by he and his colleagues was taken into account, the obligation for theatre work would be fulfilled by one or two theatre lists.

Dr K. K. Dutt (*Birmingham*) asked whether it was possible to vary the 9/11ths contract except by agreement. Dr J. S. Gibson (*Leeds*) informed the meeting that he had discussed this topic with the BMA and if the 9/11th contract had been adopted as a voluntary option, the contract holder could voluntarily change to a different sort of contract.

Dr A. G. H. Cole (*Leicester*) expressed great concern at the potential destruction of nationally agreed guidelines for pay and conditions of service. Dr D. A. Young (*Bromley*) raised the problems associated with on-call coverage of intensive care units. The Chairman, noted that the NHD calculation of such coverage must be done on the basis of work actually done. Dr Margaret L. Heath (*Lewisham*) felt that the Association should adhere to the principle of only listing commitments as fixed if they involved others. Dr E. B. Lewis (*Hythe*) expressed sympathy with the concerns of Drs Cole and Heath. Job plans weren't designed to identify work not being done, or to pay the clinician more. The object of the exercise on the part of management was to see what services were being provided, and to see whether they wanted them. If a consultant didn't have a 'standard' work week, management should be told. In closing this session, Dr MacRae expressed the hope that nationally agreed guidelines for pay and conditions of service would be maintained.

## Open forum 2

### *Changes in ODA training*

Dr Margaret L. Heath (*Lewisham*) reviewed the current activities of the National Health Service Training Authority working group on ODA training. The aim was to introduce one to one training as a less formal method of

vocational training. She informed the meeting of the way in which standards and performance criteria are to be applied. Handbooks and training guidelines would be available and it was intended to implement the new scheme by August 1991. Regional briefings would be held on the new scheme which, it was hoped, would lead to more opportunities for ODAs to share work with nurses.

### *Study leave*

Dr M. R. Bryson (*Newcastle upon Tyne*) recalled a previous meeting had considered study leave regulations. The responsibility for study leave had now been devolved to local level and he expressed concern that study leave budgets would not be protected in the face of local cut-backs. He stressed that study leave was a contractual right, and that funds must be made available to make up any shortfall. He urged Linkmen to monitor the situation closely and report any problems to the Association. Dr P. S. Gadgil (*Barnsley*) noted a problem had occurred when an SHO had applied for leave to attend the JAG meeting. This had been refused on the grounds that this was a 'social event'. The Honorary Secretary reported that on this occasion he had phoned the health authority and the embargo had been withdrawn.

Dr P. Morris, Vice President, informed the meeting that study leave was always considered during hospital accreditation visits. Most health authorities provide appropriate levels of help although there were occasional problems in London. With regard to clinical assistants there were problems as there was no provision for study leave in their terms and conditions of service. Despite this about half the health authorities granted study leave and expenses to clinical assistants. Dr Vanda Boyd (*Rotherham*) reported difficulties obtaining study leave for clinical assistants. Dr Morris felt sure it was possible to overcome this with pressure upon the authority.

### *Training for overseas doctors*

Dr D. A. Saunders, Bernard Johnson Postgraduate Adviser, College of Anaesthetists, outlined the methods available for overseas doctors to obtain training in Great Britain. Essentially, there are two ways of obtaining limited registration, with PLAB, or with a PLAB exemption. For the latter the candidate has to provide a satisfactory curriculum vitae, a job offer and the signature of 'someone in authority'. The second method of entry is through the College of Anaesthetists overseas doctors training scheme (ODTS). The ODTS has lots of applicants, but few jobs to offer. He informed the meeting that the whole system of placing overseas doctors in training posts was under review.

Currently Dr Saunders said he was placing 2 to 3 overseas doctors per week. Doctors who have failed PLAB are unable to qualify for a post through this route. In the future, Dr Saunders believed that by taking overseas doctors as SHOs and training them to the required standards, it will be possible to fill the visiting registrar posts. He hoped that it would be possible to set up a bulletin board to match posts and suitable candidates. Professor P. Hutton (*Birmingham*) emphasised the requirement for the overseas doctor to be able to communicate in good written and spoken English. He noted that problems had occurred with this in the past and he drew attention to these requirements in relation to sponsorship for PLAB exemption. The sponsor must be aware of the obligation to ensure that communication in English is adequate.

*New consent form*

Dr P. V. Scott (*Bromsgrove*) discussed the new consent form which the Department of Health wished to introduce at the beginning of 1991. Entitled 'Patient Consent to Examination and Treatment', (HC(90)22), it came with explanatory notes and had been exceedingly difficult to acquire. Dr Scott expressed his grave concern about Appendix A1, the model consent form. He was of the opinion that a single statement concerning both surgery and anaesthesia could not be signed properly by any one doctor. He highlighted areas of concern particularly where it appeared that the consent form did not allow for a change of technique on the part of the anaesthetist once consent had been obtained.

Dr D. P. Cartwright (*Derby*) expressed great dismay at the wording of the proposed consent form, but noted that consent forms can be based upon this model, leaving room for possible change. Dr Shirley Firn (*Wakefield*) felt the speed of introduction was unfair. Her Health Authority had been most reluctant to provide copies to permit study locally. Dr D. Wood (*Durham*) drew attention to potential difficulties with training paramedics and other non-medical staff if the opt-out clause was used frequently. It was noted that further advice on the training of medical students was to be provided at a later date. Dr R. L. Marks (*York*) informed the meeting that a local legal opinion had felt that the new form implied that consent would have to be obtained by a consultant. Dr R. L. Hargrove (*Westminster*) felt this was unlikely to be the case. Dr D. S. Arthur (*Glasgow*) enquired whether consent would be valid only if a full explanation of the hazards and risks had been given. He foresaw many problems if the consent form was introduced as it was at present.

Dr Scott proposed that district general managers should be told not to print the new forms, that the old consent forms should continue to be used, and the new forms should be discussed locally by all appropriate advisory bodies. A national consensus was required to produce a new consent form. This was not a matter that could be forced through; the Association and the College should make a statement to this effect. The Chairman thanked Dr Scott for his vigilance and said that this matter would be brought as a matter of urgency to the next meeting of the Advisory Committee.

*Training of paramedics*

Dr D. A. Young (*Bromley*) expressed his concern about obtaining consent for paramedic training in intubation and other skills. He felt this was especially relevant after the previous discussion. He had contacted one of the defence societies who believed that it was permissible to train paramedics during routine work and that verbal consent from the patient would be sufficient for this purpose. In his discussion with the defence society Dr Young had suggested a form of words suitable for the purpose of obtaining consent for paramedic training, as he was unsure whether verbal consent was sufficient. He sought the opinion of the meeting. Dr P. J. F. Baskett explained how he approached the problem. He did not differentiate between paramedics and medical students for training purposes and believed that verbal consent was quite adequate. Dr Stephanie Greenwell (*North Tyneside*) felt that careful choice of words such as 'assisting with treatment' would help in these circumstances.

Dr R. S. Vaughan (*Cardiff*) described local arrangements for training non-medical personnel and emphasised that health authority were legally and financially responsible for incidents arising during training of this nature. Dr P. Morris (*Manchester*) reminded members that a previous communication from the Association had stated that health authorities must know and agree to paramedic training. Dr B. Dennison (*Middlesbrough*) drew attention to the successful use of a special paramedic consent form. Dr D. P. Cartwright (*Derby*) noted the opt-out clause in the new consent form and Dr R. L. Hargrove (*Westminster*) reminded members that if verbal methods were being used to obtain consent for paramedic training, they would be totally invalid if premedication or other drugs had been administered before the verbal consent was obtained.

In closing the meeting, the Chairman expressed gratitude to the speakers for their valuable presentations. He thanked Linkmen for their constructive comments and their continuing support of the Association in maintaining the vital links between members and Council. The meeting was closed.

## Hazard Notice

### **Breathing system components—Gibeck Humidifiers HC(Hazard)(91)8**

The plastic connector used with Gibeck Humidifiers, Humid-Vent Mini and 2S Flex, may be faulty and have a moulded membrane inside the connector. This results in complete obstruction to gas flow to the patient. Products with the following batch numbers/date identification should be withdrawn immediately: all batches manufactured between January 1990 and November 1990 (90 Jan 30 to 90 Nov 94 inclusive). The code is shown on the individual product pack and carton. All such batches should be returned to Rusch UK Ltd.

### **Bionica MDS 110 PCA (Patient controlled analgesia) ambulatory syringe pump: Withdrawal from use HC(Hazard)(91)9**

A near fatal incident occurred in which a Bionica MDS 110 PCA pump infused the entire contents of a 10 ml syringe. The pump operated correctly, but the plunger of the 10 ml syringe was not positively secured to the pump carriage. This problem was previously highlighted in HC(Hazard)(88)9. There is currently no UK supplier and modifications to secure the plunger cannot be implemented without manufacturer's representation in the UK.

## Safety Action Bulletin

### **Check list for anaesthetic machines (SAB(91)10)**

Anaesthetists are reminded of the Association's booklet *Check list for anaesthetic machines* published in July 1990. These are available from the Association's offices price £3.00 (£1.50 for members).

### **Anaesthetic and respiratory equipment (SAB(91)11)**

This contains supplementary advice to that contained in SAB(89)78 on the use of 22 mm breathing system connections. SAB(89)78 advised on the publication of a revised BS 3849 which redefined the male/female sequence

of fittings, thus reducing the number of components and improving the security of 22 mm connections in breathing systems.

### **Fuel cell type oxygen analysers/monitors in breathing systems: location of the sensor (SAB(91)12)**

Oxygen analysers operating on the fuel cell principle which are connected between anaesthetic machines and lung ventilators may give an inaccurate higher reading. These analysers should be located as near as possible to the patient and, if possible, downstream of all other items of equipment.



# THERE IS NO BETTER WAY TO STAY INFORMED

**1**

**Top authorities  
systematically review  
all advances**

**2**

**Experts select  
and evaluate  
key papers**

**3**

**Comprehensive bibliography  
lists all papers  
in each area**

## Current Opinion in ANAESTHESIOLOGY

Reviews of all advances · Evaluation of key references  
Comprehensive listing of papers

Vol 4 · 1991 · No 1

No 1	<b>THIS ISSUE</b> Cardiovascular physiology and disease · P Barash Cardiac anaesthesia · NP de Bruijn Cardiovascular pharmacology · C Prys-Roberts	February
No 2	Intensive care · M Lamy · Resuscitation · N Bircher	April
No 3	Obstetric anaesthesia and analgesia · TA Thomas Paediatric anaesthesia · J Lerman Anaesthesia and medical disease · BR Brown	June
No 4	Drugs in anaesthesia · M Wood Anaesthetics · D Lundberg · Opioids · PS Sebel Muscle relaxants · WC Bowman	August
No 5	Neurosurgical anaesthesia and neurological disease · W Fitch Regional anaesthesia · F Bonnet Pain therapy · P Raj	October
No 6	Practical anaesthesia · BF Horan Monitoring and equipment · A d'Hollander Pulmonary aspects of anaesthesia · JF Nunn	December

CS  
CURRENT  
SCIENCE

### Editors

C Prys-Roberts

J G Reves

**NO RISK GUARANTEE:** You may cancel your subscription any time within 30 days of receiving your first issue and receive a full refund

#### ORDER FORM

#### Current Opinion in ANAESTHESIOLOGY

Please enter my subscription to Volume 4, 1991 (6 issues):

☐ **Personal** - from personal funds ONLY £75/\$145

☐ **Institutional** £150/\$255

Subscription rates include airspeed delivery  
Subscriptions run on a calendar year basis

☐ 30% discount for Students/ Housemen/ Residents  
Deduct 30% from the prices above and provide proof of status

Please send me further information on:

☐ Current Opinion in ANAESTHESIOLOGY DATABASE

☐ The Annual of Cardiac Surgery

☐ Current Opinion in Radiology

USA & Canada subscriptions to: Current Science, 20 North 3rd Street, Philadelphia, PA 19106-2113, USA (USA & Canada subscriptions ONLY)

Rest of the world subscriptions to: Current Science Ltd, Subscriptions, 34-42 Cleveland Street, London, W1P 5FB, UK

#### METHOD OF PAYMENT

☐ Am Ex ☐ VISA ☐ MasterCard Amount payable .....

Card no ..... Exp Date .....

Signature ..... Date .....

☐ Cheque/Eurocheque enclosed payable to Current Science Ltd

☐ Bank transfer (Details available on request)

Payment may be made in US\$ at the current exchange rate  
& sterling must be drawn on a UK bank, US\$ on a US bank

☐ Please send an invoice (institutional subscriptions ONLY)

Name .....

(BLOCK CAPITALS PLEASE)

Address .....

Zip/Postcode ..... Country .....

Return this form together with payment to address below EX

# The World's Premier Medical Publisher

## EPIDURAL & SPINAL BLOCKADE IN OBSTETRICS

Edited by

**Felicity Reynolds**, *Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.*

This book has evolved from the Obstetric Anaesthetist's Association meeting in the UK in September 1989. Unlike other books of proceedings however, the well-known contributors were invited not only to speak but also to provide a chapter reviewing their subject, prepared especially for the book.

Edited by Dr. Reynolds, the result is an interesting blend of a review of the last ten year's developments coupled with presentations of current research and coloured throughout with stimulating, amusing discussion.

- Includes unique illustrations, using state-of-the-art imaging techniques, of the anatomy of the epidural space.
- Features excellent reviews of the rapidly expanding fields of Caesarean section and the use of spinal opioids.

The closing section, The Recipient, discusses the effects that conduction blocks may have on the baby, and reviews the lay literature covering consumer's views of epidural practice.

### CONTENTS

The Epidural Space: Anatomy and Approach • Indications and Contraindications to Epidural Blockade • Test Doses • Top-ups Versus Continuous Infusion • Management of the Second Stage of Labour • Local Anaesthetic Toxicity • Taps and Patches • Neurological Complications of Epidural and Spinal Blockade • Regional or General Anaesthesia for Emergency Caesarean Section • Spinal and Epidural Blockade • Chairman's Responses • Epidural Opioids in Labour • Epidural Opioids for Caesarean Section • The Effects of Regional Analgesia • Public Opinions • Summary and Conclusions • Index.

**0 7020 1401 X ca 250pp Ills Pb**  
**August 1990 Baillière Tindall £17.50**

## ESSENTIALS OF ANESTHESIOLOGY 2/e

**David C. Chung**, *Consultant, Dept Anaesthesiology, The Mississauga Hospital, Ontario, Canada;*

**& Arthur M. Lam**, *Assoc. Prof., Dept Anaesthesiology, Harborview Medical Center, Univ. Washington, Seattle, USA.*

A primer in anaesthesia written for those about to specialise in the field, this book provides concise, yet thorough coverage of the scientific basis and the principles of safe practice. All chapters have been revised to incorporate new drugs, equipment and medical conditions.

Includes:

- Revised and expanded section on management of postoperative pain
- New section on quality assurance and risk management
- New chapter on AIDS and the anaesthetist
- New section on autologous blood transfusions

**0 7216 3084 7 304pp 50 ills SpPb**  
**1989 W.B. Saunders £15.50**

## TEXTBOOK OF PAEDIATRIC ANAESTHETIC PRACTICE

**E. Sumner & D. Hatch**

**0 7020 1336 6 560pp 103 ills Hb**  
**1989 Baillière Tindall £37.50**

## INTENSIVE CARE: A Concise Textbook

**C.J. Hinds**

**0 7020 1150 9 387pp 109 ills Pb**  
**1986 Baillière Tindall £15.95**

## OBSTETRIC ANAESTHESIA & ANALGESIA 3/e

**D.D. Moir & J. Thorburn**

**0 7020 1154 1 390pp Ills Hb**  
**1986 Baillière Tindall £23.00**

## DIFFICULTIES IN TRACHEAL INTUBATION

**I.P. Latta & M. Rosen**

**0 7020 1096 0 192pp 150 ills Hb**  
**1985 Baillière Tindall £19.95**

**W.B. Saunders & Baillière Tindall**

**HARCOURT BRACE JOVANOVICH**

Available from your bookseller. For more information please contact: The Marketing Dept, Harcourt Brace Jovanovich, 24-28 Oval Rd, London NW1 7DX, UK.

OR phone 081 300 3322

Prices and specifications may vary without notice.

THE ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND

APPLICATION FOR MEMBERSHIP

to

The Association of Anaesthetists of Great Britain and Ireland  
9 Bedford Square,  
London WC1B 3RA, England

(This application will be considered by Council at its next meeting. NO MONEY should be sent in the meantime. After acceptance of the application by Council, the Hon. Secretary will notify the applicant of Council's decision and the amount of subscription payable in the current financial year.)

.....19

To the Honorary Secretary,  
Association of Anaesthetists of Great Britain and Ireland

I (full name) ..... offer my name as a candidate

BLOCK LETTERS

for (a) ORDINARY MEMBERSHIP (b) OVERSEAS MEMBERSHIP (c) ASSOCIATE MEMBERSHIP (d) TRAINEE MEMBERSHIP of the Association (please delete categories not applicable)\*

I hold the following appointment(s) as an anaesthetist (please state grade if in the National Health Service, and indicate the approximate proportion of time spent in the practice of anaesthesia):

.....  
.....  
.....

Qualifications (with dates).....  
.....

(Signed).....

Address.....

.....Hospital Tel. No:.....

.....Home Tel. No:.....

We, being Members† of the Association, nominate as a candidate for ORDINARY MEMBERSHIP/OVERSEAS/ ASSOCIATE/TRAINEE MEMBERSHIP

.....

1. (Signed).....

(NAME IN BLOCK LETTERS)

Address.....

BLOCK LETTERS

2. (Signed).....

(NAME IN BLOCK LETTERS)

Address.....

BLOCK LETTERS

\* See the reverse of this page for details of membership categories.

† One sponsor must be an Ordinary Member of the Association.

**THIS PAGE CONCERNS YOU IF YOU ARE**  
**A MEDICALLY QUALIFIED ANAESTHETIST**  
**IN GREAT BRITAIN AND IRELAND, OVERSEAS OR**  
**IN TRAINING**  
**AND ARE NOT YET A MEMBER OF THE**  
**ASSOCIATION OF ANAESTHETISTS**  
**OF GREAT BRITAIN AND IRELAND**  
**OR IF YOU ARE A MEMBER OF ANOTHER**  
**PROFESSION WHO PRACTISES IN ANAESTHESIA**  
**AND YOU WISH TO APPLY FOR ASSOCIATE MEMBERSHIP**

**Ordinary Membership** (*United Kingdom and Ireland only*)

Voting membership of the Association (the only effective body representing anaesthetists' interests in non-academic matters).

The right of attendance at the Annual General and Scientific Meetings and all other meetings of the Association of Anaesthetists.

**Overseas and Associate Membership**

The right of attendance at the Annual Scientific Meeting and other meetings of the Association.

**Trainee Membership** (*United Kingdom and Ireland only*)

Voting membership of the Association.

Representation on the Council of the Association. The Chairman and Secretary of the Trainee Group sit on Council as voting members.

Attendance at the Annual Scientific Meeting of the Association and at the special Annual Scientific Meeting of the Anaesthetists in Training, at which Trainee Members of the Association are given preference.

**Membership Fees** (inclusive of the journal *Anaesthesia*)

*Ordinary Members:* Subscription **£100** (Husband and wife **£75** each)

*Overseas and Associate Members:* Subscription **£100**

*Trainee Members:* Subscription **£52** (**£70** after 3 years if still in training)

*Retired Members:* who receive *Anaesthesia* **£15** (there is no subscription for those who do not).

**How to apply for membership**

Fill in the application form on the other side of this page.

Have it countersigned by two members of the Association, one of whom must be an Ordinary Member.

Tear out the page and send it to the address indicated overleaf.

**Do not send money with your application**

# Contents: Anaesthesia, vol. 46, no. 7, July 1991

## EDITORIAL

- The pill, HRT and postoperative thromboembolism: cause for concern?  
*E.M. Whitehead and M.I. Whitehead*

521

## ORIGINAL ARTICLES

- Cost of intensive therapy. A description of methodology and initial results

*S. Ridley, M. Biggam and P. Stone*

523

- Immunological disturbances in anaesthetic personnel chronically exposed to high occupational concentrations of nitrous oxide and halothane

*M. Perić, Z. Vraneš and M. Marušić*

531

- Morphine compared with diamorphine. A comparison of dose requirements and side-effects after hip surgery

*S.L. Robinson, D.J. Rowbotham and G. Smith*

538

- A double-blind study of the speed of onset of analgesia following intramuscular administration of ketorolac tromethamine in comparison to intramuscular morphine and placebo

*A.S.C. Rice, J. Lloyd, C.G. Miller, R.E. Bullingham and G.M. O'Sullivan*

541

- Intubation through the laryngeal mask. A technique for unexpected difficult intubation

*M. L. Heath and J. Allagoin*

545

- Epidural bupivacaine dilution for labour. A comparison of three concentrations infused with a fixed dose of fentanyl

*H.A. Noble, G.R. Enever and T.A. Thomas*

549

- Intra-operative patient-controlled sedation. Comparison of patient-controlled propofol with anaesthetist-administered midazolam and fentanyl

*G.A. Osborne, G.E. Rudkin, M.J. Curtis, D. Vickers and A.J. Craker*

553

## CASE REPORTS

- Fatal theophylline poisoning with rhabdomyolysis. A potential role for dantrolene treatment

*M.J.A. Parr and S.M. Willatts*

557

- Epidural analgesia in Eaton-Lambert myasthenic syndrome. Effects on respiratory function

*S. Sakura, Y. Saito, M. Maeda and Y. Kosaka*

560

- Chemical meningism after lumbar facet joint block with local anaesthetic and steroids

*S.J. Thomson, D.M. Lomax and B.-J. Collett*

563

## APPARATUS

- Tracheal tube resistance and airway and alveolar pressures during mechanical ventilation in the neonate

*T.R. Tipping and M.K. Sykes*

565

- Percutaneous dilational tracheostomy. A bedside procedure on the intensive care unit

*A. Bodenham, R. Diamant, A. Cohen and N. Webster*

570

- The effect of micropore filters on the aspiration test in epidural analgesia

*G.A. Charlton and E.G. Lawes*

573

## FORUM

- Clinical evaluation of a spinal catheter technique in femoro-popliteal graft surgery

*L.M.M. Morrison, J.H. McClure and J.A.W. Wildsmith*

576

- Assessing intravenous cannulation and tracheal intubation training

*M.E. Wilson*

578

- Nausea and vomiting with use of a patient-controlled analgesia system

*S.L. Robinson and D. Fell*

580

- Incidence and management of airway problems in the CHARGE Association

*C.G. Stack and R.K.H. Wyse*

582

- Extradural vein puncture—an avoidable complication

*D. Mannion, R. Walker and K. Clayton*

585

## CORRESPONDENCE

588

## OBITUARIES

604

## INTERNATIONAL CONGRESS CALENDAR

604

## THE ANNUAL CONFERENCE OF LINKMEN OF THE ASSOCIATION OF ANAESTHETISTS OF GREAT BRITAIN AND IRELAND, 1990

606

## HAZARD NOTICE

612

## SAFETY ACTION BULLETIN

612



0003-2409(199107)46:7-8

## Clinical issues in agent vaporization technology.

New clinical techniques and vaporizer technologies now make volatile anaesthetic agent delivery more predictable and much safer. However, older vaporizers remain in use and, while sometimes offering features anaesthetists may still find desirable, they may not be equipped with key features that are available on today's units.

The debate—use of older vaporizers versus newer units—centres on several clinical issues:

- **Adverse patient reactions** can be greatly reduced by combinations of current vaporizer technologies, including: *interlocking* to help prevent activation of multiple units and delivery of agent "cocktails;" *keyed filling* to help assure filling of vaporizers with the proper agent; and *circuit isolation* to protect the gas stream from trace agent contamination. Together, these systems address hypotension, bradycardia, prolonged emergence, cardiac arrest; malignant hyperthermia and halothane hepatitis caused by unintended agent delivery.

**Malignant hyperthermia** is a pharmacogenetic complication of anaesthesia<sup>2</sup> with relaxants and inhalation anaesthetics<sup>3</sup>. Though rare, it remains a cause of anaesthetic-induced death<sup>4</sup>. It has been reported that trace agent delivered by vaporizers not fully isolated from the gas stream when "off" has initiated malignant hyperthermia in a susceptible patient<sup>5</sup>.

**Halothane hepatitis** is also related to agent delivery. The minimum initiating dosage is not known, nor is there clear indication whether it is dose- or threshold-limited<sup>6</sup>. As with malignant hyperthermia, trace halothane may present a hazard to susceptible patients, and isolation of the vaporizer from the gas stream is recommended<sup>7</sup>.

- **Dosage specificity** is enhanced, in part, by vaporizer keyed filling that helps ensure that a vaporizer is filled with the proper agent. Coupled with

vaporizer interlocking, vaporizer agent-specificity and enhanced vaporizer labelling, keyed filling helps protect against delivery of an unplanned mixture.

- **Environmental issues**—especially the growing concern about the long-term effect of agent on the surgical staff—are addressed through a variety of means associated with vaporizers:

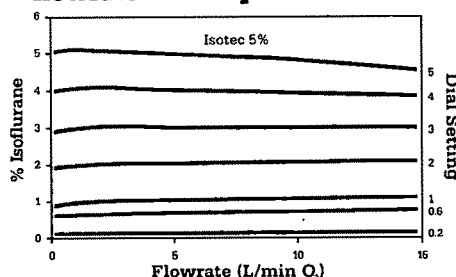
**Keyed filling:** Data are inconclusive, but studies point to keyed ports as a way to reduce pollution during filling<sup>8</sup>, especially compared to the spillage possible with funnel-fillers.

**Vaporizer mounting:** Demountable vaporizers allow removal for filling outside the operating theatre, under a fume hood or in an area with greater ventilation than the operating theatre.

**Vaporizer venting:** Some vaporizers vent excess pressurized agent to the atmosphere, increasing its concentration in the operating theatre. Current designs like the Tec 5 do not require such pressure and agent venting.

- **Dosage accuracy** is enhanced by modern vaporizers which are designed to deliver consistent and predictable concentrations regardless of flow, carrier gas composition, temperature, atmospheric pressure and backpressure from downstream components.

### Performance characteristics of the Tec 5 Vaporizer: The effect of flowrate\* on output.



\*At 22°C with oxygen flowing. Performance is similar with enflurane and halothane models.

Accuracy is also affected by control system performance, with units now offering more finite control. Advanced technology allows delivery verification through sophisticated agent monitoring.

- **Operational issues** have their greatest impact on the clinician as they affect equipment service, equipment utilization and, ultimately anaesthesia department budgets. Newer vaporizers, with their extended service and warranty plans, help reduce budget and downtime. These units can be quickly interchanged on the anaesthesia machine, allowing the anaesthetist to change agent combinations as needed.

**For more information** on these important clinical issues, and others involved in volatile anaesthetic agent vaporization, please consult an Ohmeda representative.

**Ohmeda.**

**A world of choice in anaesthesia.**

<sup>1</sup>Whitcher C: Monitoring of Anesthetic Halocarbons; Self-Contained ("Stand-Alone") Equipment. *Seminars in Anesthesia*, 5:213-224, 1986.

<sup>2</sup>Peter J: Malignant Hyperthermia, *Principles and Practice of Nurse Anesthesia*, Waugaman WR, Rigor BM, Katz LE, Garde JF and Bradshaw HW (eds), Appleton & Lange, 585-599, 1988.

<sup>3</sup>Berkow R and Fletcher AJ (eds): *The Merck Manual of Diagnosis and Therapy*, Fifteenth Edition, pg. 2455. Merck Sharp and Dohme Research Laboratories, Rahway NJ, 1987.

<sup>4</sup>Op cit Peter

<sup>5</sup>Ellis FR, Clarke IMC, Modgill EM, Appleyard TM and Dinsdale RCW: New causes of malignant hyperpyrexia. *Br. Med. J.*, 1:575, 1975; in Robinson JS, Thompson JM and Barratt RS: Inadvertent contamination of anaesthetic circuits with halothane. *Br. J. Anaesth.*, 49:745-753, 1977.

<sup>6</sup>Robinson JS, Thompson JM and Barratt RS: Inadvertent contamination of anaesthetic circuits with halothane. *Br. J. Anaesth.*, 49:745-753, 1977.

<sup>7</sup>Ibid.

<sup>8</sup>Davies JM, Strunin L and Craig DB: Leakage of volatile anaesthetics from agent-specific keyed vapourizer filling devices. *Can. Anaesth. Soc. J.*, 29:5:473-475, 1982.

# Ohmeda

Ohmeda  
71 Great North Rd  
Hatfield Hertfordshire  
England AL9 5EN  
0707 263570

A Division of BOC Health Care



A BOC Health Care  
Company  
Critical Care  
Worldwide

